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GUEST EDITORIAL

Voluntary Health Insurance

THE growth of prepayment insurance against the cost of hospitalized illness, including the cost of medical and surgical care in hospitals, has been amazing. At the present time about ninety-two million people carry some form of insurance against the cost of hospitalization. There are seventy million who carry coverage for surgical care in hospitals and approximately thirty-five million for medical care in hospitals. The growth of this form of insurance has been so rapid that we have not had time to consider certain weak points in the program. These weaknesses have become more obvious as insurance has covered more groups and has become more comprehensive. It is desirable, of course, to further extend voluntary prepayment insurance both from the standpoint of covering a still larger segment of the population and of increasing benefits offered to the individual subscribers.

The ultimate objective of voluntary prepayment insurance is to cover practically all of the insurable people in the United States for the major part of the cost of their medical care. If it failed in this objective it will be because of abuses on the part of the patients or of the doctors. It is necessary for the physician to help conserve the resources of the prepayment plan by proper limitation on his charges, by expediting the patient's course in the hospitals and by care in avoiding the over-use of hospital facilities either in terms of days of hospital care or in the use of the diagnostic and therapeutic services provided by the hospitals. If these services are over-used there is either a break down of the service and consequent poor care for everyone, or the premium rate on that type of insurance goes up. The physician should be diligent in impressing upon the patient that he is a stock holder in a mutual company and it is not only his obligation but to his own advantage to conserve the resources of the company.

Rising premium rates due to excessive use or other abuses will price voluntary sickness insurance out of the market. We can rest assured that if abuses prevail and that, as a result, the rates are too high or coverage is inadequate, some form of government subsidy or even compulsory insurance will be forced upon us. We as physicians are in a better position than any other group to control over-use and to educate our patients in the importance of conserving a valuable asset. We are also morally obligated to control abuses that arise within our own ranks.

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EDITOR'S NOTE: Dr. Walter B. Martin is of Norfolk, Virginia, and President-elect of the American Medical Association.

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CARDIOVASCULAR EMERGENCIES, THEIR DIAGNOSIS AND TREATMENT*

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The prompt and correct diagnosis of cardiovascular emergencies and their proper treatment are among the most common problems confronting the general practitioner. In discussing this problem with you tonight, I shall try to deal with the situations as it has been my custom in the private practice of cardiology. In some instances where my experience has been rather limited, I shall rely upon the opinion of those who have had greater experience with a given method or a specific drug.

The arrhythmias are perhaps the most common cause of acute concern in patients with and without cardiac disease. Even patients who have normal hearts, and who are generally emotionally quite stable, may be upset and frightened by the occurrence of an arrhythmia and there may be symptoms, such as precordial aching or severe pain, vertigo, syncope, vomiting, and at times, congestive failure, if the heart is extremely rapid over a long period of time. Of course, the most serious disturbance of the circulation occurs with the ectopic arrhythmias with rapid rate—the paroxysmal tachycardias—and the time of the change from one mechanism to another, as from a tachycardia to a complete heart block, when there might be a ventricular standstill of varying lengths of time.

The paroxysmal tachycardias include paroxysmal auricular flutter, paroxysmal auricular fibrillation, auricular, nodal and ventricular tachycardia and ventricular fibrillation. Any of these tachycardias may revert to normal sinus rhythm within a short time or they may persist for hours, or at times days, thereby causing severe disturbance in the circulatory dynamics and perhaps death due to congestive heart failure, or coronary insufficiency, or myocardial ischemia. According to Henderson and others, a change from a rate of around 80 to 130, usually increases the minute volume output, for, despite the fact that ventricular and stroke volume may be decreased, the product of heart rate and stroke volume is increased. When the rate is between 130 and 180, however, no further increase in minute volume occurs, and with further increased cardiac rate, total minute volume is actually reduced. This may result in cerebral anoxia, fainting or convulsions, as well as myocardial ischemia mentioned above. If the tachycardia persists, there may be further decrease in cardiac output with shock and failure. One interesting example of this was described quite a few years ago by Paul White and me, i.e., cases of status anginosus, resembling coronary thrombosis, but precipitated by paroxysmal tachycardia and relieved as soon as the paroxysmal tachycardia was controlled; our cases had the usual Heberden's angina of effort between attacks of tachycardia. During, and at times for hours after, an attack of paroxysmal tachycardia, the electrocardiogram may show the typical RS-T and T wave changes of coronary insufficiency. In fact, this is a wonderful test of the sufficiency of a patient's coronary arteries.

The supra ventricular tachycardias, auricular or nodal tachycardia and auricular fibrillation and auricular flutter, may occur in a normal or an abnormal heart. They may be precipitated by emotional disturbances, especially sudden ones. These arrhythmias occur most often in association with rheumatic or arteriosclerotic heart disease, and a recent study points out that the basal metabolic rate is normal in over 85% of cases of supra ventricular tachycardia. Ventricular tachycardia almost always is an indication of organic heart disease and is not an infrequent complication of myocardial infarction and adds gravity to the prognosis.

Since in many cases of supra ventricular tachycardia, the rhythm will revert to normal after a relatively short time, it is not always necessary to institute treatment at once and simple reassurance may be all that the patient needs. All cases should be carefully watched, however, for failure or shock may develop rather suddenly. I believe it is wise to start therapy in cases of ventricular tachycardia as soon as a definitive diagnosis is established, for, as already noted, this type of tachycardia almost always occurs in patients with organic heart disease and further strain on the already damaged heart may be harmful or even disastrous.

*Read in part before the annual meeting of The Medical Society of Virginia at Roanoke, October 18-21, 1953.

Certain general measures may be of value in the treatment and prevention of tachycardias. These are rest, sedation and the avoidance of tobacco, alcohol, caffeine and emotional upset. I have one physician friend-patient, who used to go to football games and perhaps indulge in all of the above, and then come home and have paroxysmal auricular fibrillation, which, fortunately, was quickly controlled by quinidine. I am sure that there are many people who would rather risk an occasional bout of transient paroxysmal tachycardia, than to have to abstain or refrain from some of their greatest pleasures. Over-eating or eating certain food will bring on an attack in certain individuals.

Before attempting to institute any specific therapy, a correct diagnosis, at least as to whether the tachycardia is supra or infra ventricular, is essential. I might add here that in some cases the only way to be absolutely certain is to have the help of the electrocardiogram. Unless the exact mechanism of the tachycardia has been shown to be due to ventricular tachycardia or auricular fibrillation, it is worthwhile to try to terminate the attack by pressure on the carotid sinus or eyeballs or by causing the patient to gag by means of mechanically stimulating the pharynx with a feather. Pressure over the carotid sinus is applied with the patient in the prone position and with the head turned away from the side to be stimulated. The site to be pressed is recognized as a pulsating area which is painful to pressure at the angle of the jaw. The right carotid sinus is more sensitive than the left. Pressure should be applied on first one side, then the other, not both sides at once, and not over ten to twenty seconds at the time, especially in older people; it should be stopped sooner if slowing of the heart rate occurs. Cases of syncope, convulsions and even hemiplegia have been reported following this procedure. Certain drugs, such as Mecholyl or Lanatoside C, augment the carotid sinus effect which others, such as Benzedrine, Epinephrine, Neosynephrine and large doses of quinidine may inhibit the effect.

Eyeball pressure, according to Sam Levine, stimulates the oculocardiac reflex and produces inhibitory effects upon the auricular muscle and atrial ventricular conduction. Pressure for twenty to thirty seconds is applied over both eyeballs simultaneously, the finger pressing on the closed eyes below the supra orbital ridge and not over the cornea. Some patients may respond to this method of treatment, but not to carotid sinus pressure.

AURICULAR FIBRILLATION

Auricular fibrillation is the most common arrhythmia of serious import. In this section it occurs most frequently in patients who have arteriosclerotic heart disease and, secondly, in patients with rheumatic heart disease. However, it frequently occurs in patients with thyrotoxicosis and in such circumstances it is very difficult to control the ventricular rate or revert the rhythm to normal sinus until the thyrotoxicosis has been controlled. Occasionally the strain thrown on a relatively normal heart by pneumonia and other febrile diseases or by extreme physical exertion will set off auricular fibrillation in a relatively normal heart.

While working with Sir Thomas Lewis, I thought the circus movement mechanism of auricular fibrillation was a fact, not a theory. However, when I went over to Vienna and worked with David Scherf, I became acquainted with the theory that both auricular fibrillation and auricular flutter are the results of stimuli arising in a single, exciting ectopic focus. According to the latter opinion, all auricular arrhythmias have a similar mechanism, the only difference between them being the number and frequency of impulses arising from a single focus. Printzmetal and his co-workers have thrown a great deal of support to this theory.

Paroxysmal auricular fibrillation usually starts suddenly, and the ventricular rate ranges around 130 to 170 and the rhythm is, of course, totally irregular. There is a pulse deficit and, of course, the intensity of the heart sounds varies a great deal. Practically speaking, a rhythm that on auscultation appears totally irregular, associated with a ventricular rate of above 130, may be considered as due to auricular fibrillation and treated as such, even without electrocardiographic confirmation.

There are, in general, two main methods of treatment of paroxysmal auricular fibrillation, not associated with thyrotoxicosis or active rheumatic myocarditis. The choice rests between the use of digitalis or quinidine or the combination of the two. If the case has been proven by electrocardiogram to be due to auricular fibrillation, the reason for electrocardiographic proof is to rule out rare cases of ventricular fibrillation, very frequent ventricular premature contractions alone, or combined with ventricular tachy-

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cardia, in which digitalis would be contraindicated. I personally should be inclined to use digitalis first, for, at times, the rapid ventricular rate may rather suddenly cause failure and/or shock. The above applies especially to patients with definitely demonstrable cardiac damage and to patients seen for the first time and in which the status of the cardiovascular system is uncertain.

Digitalization may be accomplished by various methods, using various types of digitalis preparation. I believe that each doctor should thoroughly familiarize himself with one standard oral method and one standard intravenous method, and then stick to one or the other of these unless some unusual situation arises.

If a patient is seen with paroxysmal auricular fibrillation and a rapid ventricular rate who has not been on digitalis and who is not in failure or shock, then I would be inclined to digitalize the patient orally, using Digitoxin, starting with milligrams 0.4, and repeating this again in four hours, and again in four to six hours, and if the ventricular rate were satisfactory, say 70 to 90 per minute, I would switch to whole leaf digitalis preparation, giving on the average of grains one and one-half daily. Digitoxin has the advantage of acting quickly even when given orally. However, it has the disadvantage of being excreted slowly and hence it is more likely to cause some untoward reaction, especially nausea and vomiting, which may persist. Some people prefer Digoxin, a glycoside of digitalis lanata. It has the advantage of being a pure substance of definite composition and is almost completely absorbed from the intestinal tract. Furthermore, it is eliminated rapidly and hence it is less apt to cause toxic effects, such as may occur with Digitoxin. The dosage required for digitalization varies a great deal from person to person and the dose must be adjusted for each individual. The digitalization dose of Digoxin varies from 1.5 milligrams to 5.0 milligrams. An initial dose of 1.0 to 1.5 milligrams may be tried and then 0.5 milligrams every six hours until the desired ventricular rate is obtained. The maintenance dose also varies from 0.25 to 0.75 milligrams daily and, again, I would prefer to switch to whole leaf digitalis.

If there were any doubt about the existence of failure or the possibility that the patient had been partially digitalized, I would use Lanatoside C (Cedilanid) intravenously. I have found this drug exhibits less toxic side effect and it is excreted fairly rapidly; furthermore, it seems to me more likely to cause certain supra ventricular tachycardias to revert to normal rhythm. The initial intravenous dose may be one-half of the total digitalization dose, i.e., milligrams 0.8, and then followed in four hours with milligrams 0.4, and in another four hours with milligrams 0.4, or in less urgent cases milligrams 0.4 may be given every four hours for four doses. In urgent cases the whole digitalization dose of milligrams 1.6 may be given at one time and I have never seen any ill effects from this.

Whole leaf digitalis may be used; however, its action is slower, and, again, I would prefer obtaining digitalization with one of the more rapidly acting drugs, and starting the patient on whole leaf maintenance dosage.

When the ventricular rate has returned to around 80, and if the auricular fibrillation persists, the use of quinidine sulphate may be considered; or it may be decided to use quinidine initially; in either case, the administration of the drug could be the same. Again, there are various methods of giving quinidine. It seems reasonable to acquaint oneself with a given method and stick to it unless contraindication arises. I usually use the following: Give the patient a test dose of quinidine, grains three, and if no untoward symptoms develop, then give quinidine sulphate, grains six, every two hours for five, six or even seven doses, if normal rhythm is not restored before. I hasten to add that I use the above procedure only on patients in the hospital and that I insist that the intern check the patient, especially the rate and rhythm at the apex, before the administration of each dose. If the patient is treated in the home, then perhaps we could give, after the test dose, grains three every three hours, and if this did not secure the desired results, we might try grains six every three hours during the day. When the rhythm has been restored to normal, the dose is reduced gradually and after a period of a week or so it may be left off; on the other hand, if there is a tendency for any arrhythmia to recur it may be necessary to continue a maintenance dose of grains three, three times a day or every four hours, or this may be increased to six to nine grains, three times a day or every four hours, depending on the individual case. In reducing dosage, I usually taper off with grains three, every two hours for a day, then

grains three, every three hours, and then perhaps grains three every four hours. Quinidine, if used properly, is a safe and good drug. If not used properly, the results will be poor and it must be remembered that in contradistinction to digitalis, quinidine has practically no accumulative effect and is rapidly excreted, so give what is needed to get results, no more and no less. The most common premonitory signs of over-dosage may be grouped under "cinchonism," and include tinnitus, deafness, headache, blurring of vision, nausea and vomiting, abdominal cramps and diarrhea. If given rapidly intravenously, there may be respiratory depression, convulsion and cardiac standstill, from which death may result. Quinidine may produce certain electrocardiographic changes, the most common being widening of the Q-T interval; this is due to widening of the R-T interval and only occasionally to widening of the QRS complex, and when the latter occurs it indicates a toxic effect and careful watch should be maintained and perhaps the quinidine discontinued.

Intramuscular quinidine has been used but I see no definite need for it in auricular fibrillation.

Pronestyl has been used with good results by some in treating acute paroxysmal auricular fibrillation, but I prefer to save it for ventricular tachycardia and will discuss it there.

If digitalis is judged to be the cause of auricular fibrillation or other tachycardia, the use of potassium chloride or acetate given orally must be considered, and it is reported to eliminate the arrhythmia within thirty minutes. It may be given in doses of two to ten grams of a 20% solution in syrup of citric acid.

The treatment of auricular fibrillation, due to thyrotoxicosis, is first the treatment of the thyrotoxicosis and then the auricular fibrillation by one of the methods described below.

Auricular Flutter

The exact mechanism of the auricular physiology in auricular flutter differs in the two schools of thought as discussed above concerning auricular fibrillation. In auricular flutter the auricular rate varies between 250 and 350 but the auricular rhythm is usually regular. The ventricular rhythm is usually regular and may vary from 125 to 180, often it is 160, and I think that a fixed rate of 160, i.e., one not influenced by position, movement, etc., is very apt to be associated with auricular flutter. The ventricular rate may vary quickly and the rhythm may change quickly due to varying degrees of auricular-ventricular block. Sometimes auricular flutter may be distinguished from auricular fibrillation by detecting regular venous pulsations in the neck at a rate of around 250. Often we have to rely upon the electrocardiogram for the final diagnosis, and if there is a complicating bundle branch block it may simulate ventricular tachycardia. Carotid sinus pressure may cause a sudden change in the ventricular rate, say from 160 to 80, i.e., exactly doubling the block, and this may return suddenly in a short time to 160. In auricular tachycardia the rate may drop from, say 200 to 70, and stay there.

The accepted treatment of auricular flutter is the use of digitalis, for, as has been stressed since Sir Thomas Lewis' original work in 1911, digitalis often will convert auricular flutter to auricular fibrillation and then the rhythm will become normal after the drug is discontinued. The method of digitalization of choice depends upon the urgency of the case and the same principles should be followed as discussed above under treatment for auricular fibrillation. If sinus rhythm does not occur within a relatively short time, say three to four days after leaving off the digitalis, then quinidine may be given orally as described above. Some cases of flutter persist despite digitalization to the point of nausea and despite full doses of quinidine. If the ventricular rate is 80, for example, because of a 4:1 block, I think the patient may be watched along and the rate and rhythm may stay the same for years.

Auricular and Nodal Tachycardia

Auricular and nodal tachycardia can be distinguished only by the electrocardiogram and sometimes not then. The treatment of these conditions is accepted as being the same. However, it is my clinical impression that auricular tachycardia will revert to normal rhythm more quickly with the usual treatment. These tachycardias do not necessarily indicate organic heart disease and are more frequently functional, and may be brought on by excessive use of tobacco or alcohol, infections, thyrotoxicosis, gaseous indigestion, emotional upset, and the like. The individual episodes may be very short lived and the patient may have three to four or seven to eight attacks of very short duration every day or so for many years. Many patients have learned

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how to stop their individual attacks by various maneuvers which stimulate the vagus nerve, for example, holding the breath, coughing, vomiting, drinking ice water, lowering the head over a bed, and even standing on the head. In trying to establish the previous existence of attacks, it is important (as in every cardiac case) to take a detailed history, with special emphasis on whether or not there was a sudden onset and sudden cessation of the rapid palpitation or fluttering of which the patient complains. There may be a sensation of irregular palpitations at the beginning and ending of the episodes in cases which have premature contractions or extra systoles at this time. The attacks usually start very suddenly with a ventricular rate of about 180 to 220 per minute. The ventricular rate is usually faster than in flutter and there is no change in rate associated with change in position, exercise, etc. The rhythm is usually absolutely regular, whereas in ventricular tachycardia there is often a very slight irregularity sufficient enough to cause slight changes in the intensity of certain sounds.

The episodes may cease quickly and if the person has a normal heart there is no great haste about trying to stop the attack. Rest and relaxation are important, a mild sedative such as Seconal may help. When first seen by the doctor it is certainly wise to try carotid sinus pressure and/or eyeball pressure as described above in detail. If these measures are not successful and if the patient is not already fully digitalized, the treatment method of choice, in my opinion, is the intravenous administration of Lanatoside C, giving 1.2 milligrams or even 1.6 milligrams. Often within ten to thirty minutes the rhythm and rate return to normal. Other methods of digitalization may be tried. If Cedilanid is not used, then quinidine sulphate may be used, again giving grains six every two hours and checking the patient before each dose.

Syrup of ipecac, four to eight cc. by mouth, will often cause vomiting and thus stop an attack. It is unpleasant.

Mecholyl (acetyl beta methylcholine) has been used to some extent and is helpful in treating supra ventricular tachycardia. I rarely use it now. It may produce severe nausea, vomiting, diarrhea, severe precordial pain and occasionally collapse and shock, and deaths have been reported. It is given subcutaneously in doses of twenty milligrams and this may be repeated in twenty to thirty minutes. It is always imperative to have atropine sulphate, grains 1/60 to 1/30, to be given intravenously for relief of the serious untoward symptoms. Acetyl-choline is a similar drug with less side effects. Neostigmine given in doses of 1.0 milligrams intramuscularly is also a useful drug for this purpose.

Neosynephrine given intravenously in doses of 0.5 to 1.0 milligrams reportedly often stops an attack within ten to thirty seconds. It produces a rise in blood pressure, stimulation of the cardio inhibitor fibers in the aortic arch and carotid body and reflex cardiac slowing. The blood pressure usually returns to normal in ten minutes. Obviously it should not be used on patients who have a high blood pressure during the attack.

Pronestyl has also been used and is occasionally helpful.

VENTRICULAR TACHYCARDIAS

Ventricular tachycardia, of these ectopic paroxysmal ventricular tachycardias is, of course, the most common; ventricular fibrillation, at least recognized as such clinically, is rather rare. However, it is believed that terminal ventricular fibrillation is frequent as a cause of death, particularly following myocardial infarction. Whereas the supra ventricular tachycardias are often functional, ventricular tachycardia usually is indicative of, and associated with, severe organic heart disease. It is not an uncommon complication of myocardial infarction and may occur under various conditions in people with organic heart disease, for example, marked physical strain, pneumonia and other febrile diseases. The onset is usually sudden, although the electrocardiogram beforehand may show frequent or short runs of ventricular premature contractions. The rhythm is practically regular but may not be absolutely regular at times. The rate varies from 140 to 180 to 200, usually nearer 200, and the rate is not changed by position, etc. The symptoms, in addition to apprehension, palpitations, throbbing in the neck, etc., may be those of coronary insufficiency, substernal aching and/or cerebral anoxemia and/or circulatory failure and shock. Before starting definitive and specific treatment in a patient suspected of this condition, an exact electrocardiographic diagnosis must be established, for, whereas digitalis is usually indicated in auricular or nodal tachycardia, digitalis is contraindicated in ventricular tachy-

cardia; it may initiate ventricular fibrillation in the already irritable myocardium. It must be remembered that digitalis often causes ventricular premature contractions and, if digitalis were discontinued in these cases, especially if the ventricular contractions arise from several different foci, there would be less frequent ventricular tachycardia. The mechanical measures of carotid sinus pressure and/or ocular pressure will not be helpful.

Quinidine still deserves the first trial in the treatment of ventricular tachycardia, as it usually causes the tachycardia to revert to normal when given orally. Occasionally the oral method does not stop the attack and in such cases quinidine may be given intramuscularly or intravenously; however, rather than use quinidine by these methods, I would prefer intravenous Pronestyl. Oral quinidine may be started on the grains six, every two hours, schedule, checking the patient, of course, before each administration. If the patient remains in good condition, but the attack persists for over six to eight hours, it may be necessary to give grains nine, every two hours. In the old days even larger doses were given; however, if one failed to get desired results with the above, it would be wise to switch to Pronestyl.

Procaine amide (Pronestyl) is a direct myocardial depressant. It is supplied in 250 milligrams capsules for oral use and in ampules containing 100 milligrams per cc. for intravenous use. If the oral route is used, 500 milligrams every three or four hours may be tried. However, if the attack is not stopped within a few hours, or if the patient is not doing well, I would switch to the intravencus route. When I give Pronestyl intravenously, I have a nurse or intern take the blood pressure as near as continuously possible and at the same time I have an intern or technician take the electrocardiogram, preferably on a direct writing machine; however, with practice one can watch the string shadow and guess pretty well what is going on. The change in the rhythm may be quite sudden and occur within twenty, thirty or sixty seconds after the start of the intravenous medication, or there may be further widening of the QRS complex and in either incidence the Pronestyl should be stopped at once. Pronestyl generally causes no serious reactions; however, a marked sudden drop in blood pressure may occur, resulting in temporary shock or even a cardiac standstill. Neosynephrine, one to five milligrams intramuscularly, has been recommended to counteract this reaction. Fortunately, so far I have not had to use the Neosynephrine. The Pronestyl is injected at the rate of 100 milligrams, or 1 cc., per minute, no faster, a careful watch being kept on the blood pressure and the electrocardiogram, and the whole 10 cc., or one gram, may be given in ten minutes if the attack doesn't stop and if there is no further widening of the QRS. Again, may I point out that as soon as the ventricular tachycardia ceases, the injection should be stopped. If the attack continues, the above procedure may be repeated every thirty to sixty minutes until the attack stops.

We have had very good results with Pronestyl intravenously and have used it in about fifteen cases of ventricular tachycardia and in no case had it failed to stop the attack. The results have been quite dramatic, and I feel sure that in one or two cases Pronestyl has actually been a life saving measure. One case, a man in his sixties, had had a recurrent myocardial infarction and had a ventricular tachycardia and his pulmonary oedema was getting progressively worse, and if ever a man apparently rapidly was approaching a lethal exodus, this one was. Pronestyl was given intravenously, his ventricular tachycardia reverted to normal rhythm and he rapidly improved and survived this attack.

Magnesium sulphate has been used in the past, given intravenously, ten to twenty cc. of a 20% solution. Potassium salts (e.g., postassium chloride, enteric coated tablets, in doses of 1 to 5 grams two to four times a day) have been reported to be particularly efficacious in ventricular tachycardia due to digitalis toxicity.

Ventricular fibrillation is rarely diagnosed clinically as the patient usually dies rather quickly or the rhythm changes. Clinically, it is very like Stokes-Adams syndrome due to cardiac standstill. If possible, an electrocardiogram should be obtained during the attack and, if the patient survives and has recurrent similar episodes, it is reasonable to assume that these too are due to ventricular fibrillation. Intravenous quinidine, as quinidine lactate six to nine grains, should be given at once and, although there is danger in this route of administration, the disease is often fatal and the chance worth taking. Pronestyl should be of definite help, although I personally have not tried it.

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Complete heart block with sudden failure of idioventricular pacemake and asystole is a common cause of Stokes-Adams syndrome, although a change from one rhythm to another may cause asystole and Stokes-Adams. Adrenalin is the immediate treatment of choice. In extreme cases in shock it may be given intravenously or directly into the heart. In the usual case, one-half to one cc. of 1:1,000 solution of adrenalin should be given hypodermically. This may be repeated as necessary if the attacks recur and are severe. Adrenalin in oil, one cc. may be given every twelve to twenty-four hours after the initial dose of adrenalin if the attacks tend to recur; likewise, Ephedrine sulphate, grains one-half, three to five times a day may be used.

A very interesting and perhaps far reaching paper was presented at the American Heart Association meeting in April by Zoll *et al.*, on "External Electric Stimulation of the Heart". "Electric impulses externally applied across the intact chest produced effective heart beats in humans with idioventricular rhythm and in humans and dogs with ventricular standstill. The effective electric stimuli are monophasic rectangular waves, 2 to 20 milliseconds long, and 40-150 volts in intensity. This method of external electric stimulation therefore provides an externally controlled supraventricular or ventricular pacemaker, discharging at any desired rate and behaving like a natural, intracardiac parasystolic focus. It can be used to study cardiac arrhythmias. It has also been used successfully to arouse the human heart from ventricular standstill, and to keep it beating as the sole pacemaker over a period of 5 days, until the intrinsic idioventricular pacemaker revived spontaneously".

I have not seen the machine as yet, but it is apparently rather simple and should be useful.

Time does not allow a discussion of the pathogenesis of cardiac failure, nor will I bore you with a description of paroxysmal nocturnal dyspnea (cardiac asthma) or the more severe acute pulmonary oedema.

When the patient is seen with cardiac asthma, the first thing to do is to get the patient up on the side of the bed or in a chair; this will often cause relief. If not, then I think morphine sulphate, grain ¼, and atrophine sulphate, grain 1/100, may be given by hypodermic and often there is quick relief. Aminophyllin may be give intravenously or by mouth or by suppository. Since the occurrence of cardiac asthma is evidence of left ventricular failure, I believe these patients should be digitalized or kept on a maintenance dose. If the patient is seen in acute pulmonary oedema or progresses to acute pulmonary oedema, then certainly morphine sulphate should be given intramuscularly at once, or if the pressure is low and the patient is in shock it may be given intravenously. I prefer atropine with morphine.

If oxygen is available it should be started at once, using the emergency mask and then switching to nasal oxygen or the tent. Intravenous aminophyllin grains 7 1/2 should be given at once, but administered slowly. Then if the patient has not been on digitalis some form of quick acting digitalis should be given. I prefer Cedilanid given intravenously in a full 1.6 milligram dose. Strophanthin K or Strophanthin G (Quabain) milligrams 0.25 intravenously may be used. In either case the patient should be digitalized immediately after the acute attack, the rapidity and method of digitalization depending upon the progress of the individual case. I think it is wise to use one of the mercurial diuretics intravenously, and I personally give Mercuzanthin 2 cc. intravenously as soon as the digitalis has been given. So called bloodless phlebotomy by use of tourniquets may be of help. The tourniquets are placed on three extremities using sufficient pressure to occlude veins, but not arteries. They are rotated every fifteen to twenty minutes. If the patient doesn't improve in thirty to forty minutes, and if there is no evidence of anemia, 500 to 800 cc. of blood should be withdrawn from a vein quickly and the result may prove dramatic.

Just a word about angina pectoris or paroxysmal coronary insufficiency. The treatment, of course, is nitroglycerine or, at times, amyl nitrite for the immediate episode. Prevention of attacks cannot be taken up here.

The most important thing in treating coronary occlusion with myocardial infarction is first to relieve the pain. If the pain is not severe, Demerol 50 to 100 milligrams may be given hypodermically. However, many cases require morphine sulphate 1/4 grains and again I use atropine sulphate. If this does not relieve the pain in twenty minutes, or if the pain seems to be getting worse, then the same dose may be repeated and occasionally it may be necessary to give morphine sulphate intravenously. I have found that "H.M.C." often relieves pain when morphine

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sulphate and atropine fail, or, at times, if the patient has had morphine sulphate grains 1/2, I might give hyoscine hydrobromide grains 1/200 to grains 1/100. Oxygen by emergency mask or by tent should be given certainly in all patients who continue to have pain and, in fact, I believe it is wise to give it in practically all cases. Intravenous aminophyllin grains 7 1/2 often helps relieve pain and is certainly indicated in patients who show any tendency toward left sided failure and cardiac asthma. Some doctors use quinidine routinely in cases of myocardial infarction; I don't believe this is necessary; however, if any irregularity of the heart develops, such as premature contractions, I would use quinidine; so, also, if digitalis is to be given to that particular patient. The ectopic tachyardias should be treated as previously discussed. Some time ago it was considered unwise and dangerous to give digitalis to patients who had had an acute myocardial infarction. I believe this opinion has changed and if such a patient begins to have failure and if this is not relieved immediately by a salt free diet and mercurial diuretics, it seems wise to me to digitalize the patient. I believe it is wise to give quinidine, as noted, and unless the situation is urgent, rapid methods of digitalization need not be used.

Morphine is the drug of choice for relief of severe pain of coronary occlusion, but it has certain untoward side effects at times, such as nausea and vomiting, constipation, distention and depression of the renal function. Therefore, after the initial dose, or as soon as possible, it is wise to switch to Demerol 50 to 100 milligrams. Dilaudid grains 1/12 to 1/24, is a good drug and Pantopon is liked by some.

The most serious frequent complication of coronary occlusion is shock. This shock is due to a sudden failure of the heart to pump blood into the tissue. It is a fall in cardiac output and is not due to blood loss or to decrease in venous return. This so-called cardiogenic shock results from, in part, myocardial weakness connected with the infarction. However, I feel this is not the sole cause and there are certain reflexes which are poorly understood but which nevertheless play an important part in this phenomena. This shock may occur suddenly, or there may be a drop of blood pressure over a period of several hours until it gets to 80 or below, systolic. Theoretically, transfusions or intravenous fluids are not indicated, and, in fact, may lead to further increase in venous pressure and congestive failure. Actually, however, this danger of inducing failure is not so great, as has recently been proven. Intra arterial infusions have been used, but I have not had personal experience with these. Neosynephrine in doses of 3-5 milligrams intramuscularly may be tried and repeated every two to three hours. Masters and Griffin have used ACTH and Cortisone, but without good results so far. At the present time I believe the best treatment for this type of shock is the use of Norepinephrine. Sampson *et al.* and Griffiths *et al.* reported good results with this procedure. We use essentially the same technique as Sampson, and is as follows: "The agent was diluted in 5 per cent glucose Ringer's solution, or other suitable menstruum. Usual initial dilution was 4 milligrams of the bitartrate monohydrate (Levophed) per liter. This was administered intravenously at rates varying from 4 to 80 drops (0.001 mgm. to 0.02 mgm.) per minute as necessary to maintain the blood pressure at appropriate levels. The concentration was increased to 8, 12 and even 16 milligrams per liter when adequate response was not obtained at lesser concentrations and to restrict parenteral fluid intake. Withdrawal was accomplished very gradually by decreasing the rate of infusion and often the concentration. Therapy was continued as long as necessary. The important thing in treatment of shock is time if the blood pressure of a previously normotensive drops to 100 systolic or below, or if a hypertensive drops to 110 systolic or below, and doesn't start back up in two hours treatment should be started."

Anticoagulant treatment should, in my opinion, be started on all cases of proven myocardial infarction unless there are definite contraindications in the individual patient. If a peripheral embolism occurs the treatment varies with the site of the embolus and the time that a patient is seen. Intra arterial papaverine 50-100 milligrams every four hours, given proximal to the occlusion, may be helpful. Likewise, intra arterial Priscoline, 50 milligrams every four to six hours, has been of help. Various ganglionic blocking agents have been used, e.g., C5 (bistrimethyl-ammonium pentone dibromide). I have had no experience with this. Embolectomy is the treatment of choice if a large artery is blocked and a competent surgeon can be obtained in eight to seventeen hours. Dr. Donald Daniel recently removed

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a beautiful saddle embolus at the aortic bifurcation and the patient got along beautifully and is still alive —patient of Dr. Stewart Gilman.

Just a few words about syncope which occurs in many conditions and usually in patients who do not have organic heart disease. Neurogenic or vaso depressor syncope is the most common and is produced by a reflex peripheral vasodilatation and may be brought on by fright, pain, emotional upsets, etc. The usual treatment is simply to get the patient's head below his body. If clonic twitches occur, adrenalin .5 cc. 1:1000 col. hypodermically may be needed.

A hypertensive carotid sinus may be the cause of fainting in some patients. If the diagnosis can be established, the patient should be warned against things which will stimulate the sinus—tight collars, etc.—and it is reasonable to try ephedrine sulphate, grains 1/2, three times a day, or Paredrine, 60 milligrams, three times a day, or atropine sulphate, grains 1/200, three times a day.

Postural hypotension at times causes syncope, and the treatment depends on the cause if it can be found.

Hypoglycemia may also cause syncope and the treatment depends on the cause of the hypoglycemia.

Emergency treatment may be required during the acute stage of rheumatic fever and rheumatic pancarditis, because of the occurrence of pericarditis, congestive failure and arrhythmia. The latter two have been dealt with in general. If the patient with pericarditis develops an effusion enough to cause a tamponade then a pericardial paracentesis should be done at once, and it may be life saving. Apparently ACTH and Cortisone are useful in the treatment of the acute severe stage of rheumatic fever. The exact mode of action is not known and the question as to whether or not the ultimate progress and course of the disease is influenced has not, in my opinion, been proven. Nevertheless, since these drugs apparently can tide the patient over the very acute phase, their use is worthwhile. ACTH may be given somewhat as follows: 25 milligrams every six hours for six days, then a daily dose of 60 milligrams for seven days, then 40 milligrams for two or three weeks. Cortisone in a single daily dose of 200 milligrams may be given intramuscularly, or, if it can be given orally, it is more effective and can be given 75 to 100 milligrams every six hours for about the same length of time.

Finally, hypertensive encephalopathy and crisis may occur in the course of essential hypertension, malignant phase usually, or in acute nephritis, or in pheochromocytoma. These attacks, if they persist, may at times be helped by intravenous magnesium sulphate SO4, 20 cc. of a 10% solution, or by withdrawal of spinal fluid, or by 50 cc. of 50% intravenous dextrose. Quite effective is the use of hexamethonium chloride, intravenously, starting with 5 mgm, then giving 5 mgm every two to three minutes until the desired drop in blood pressure has occurred. Another person must be on hand to record the blood pressure as the drug is given. If the blood pressure drops below the desired level, elevation of the foot of the bed will aid recoverage of adequate blood pressure levels. On the other hand if no drop occurs, the effect may be potentiated by the head up position. It must be emphasized that this is an extremely powerful drug, and must be given cautiously. If the crisis is proved to be due to a pheochromocytoma the tumor should, of course, be removed.

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THE HEART AND GALL BLADDER DISEASE*

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The purpose of this paper is to re-emphasize the apparent close association between the occurrence of coronary disease and cholecystitis, and to review briefly the evidence that impulses occurring in the biliary tract and other abdominal viscera may act as trigger mechanisms in the production of various cardiac arrhythmias and angina pectoris. It will also be pointed out that the differential diagnosis between the two conditions is often difficult because of the fact that the pain impulses may be transmitted to the same or overlapping areas in the spinal cord.

Examples of various cardiac abnormalities which improved after cholecystectomy will be given. In addition, a patient with gall bladder disease resembling angina pectoris and one with myocardial infarction resembling gall bladder disease will be described. The mortality of cholecystectomy in patients with heart disease and the probable benefits to be derived by cholecystectomy will be discussed.

The association between gall bladder disease and varicus affections of the heart has been known for many years; in fact, Osler in his *Practice of Medicine*[1] described palpitation, distress around the heart, convulsive seizures, and even fatal syncope occurring with acute gall bladder attacks. McKenzie pointed out that the famous English surgeon John Hunter, who died with angina, was found at autopsy to have gall stones.[2]

A review of some of the literature[3,4,5,6,7,8,9,10] reveals that the autopsy incidence of gall bladder disease in patients with coronary artery disease was found to vary from 17.5% to 48.8%, while only 11.5% of cases without coronary disease were found to have cholecystitis.

Clinical studies similarly show that 6.8% to 7.5% of patients with clinical coronary sclerosis (as evidenced by angina pectoris or myocardial infarction) had cholecystitis, as compared with 1.7% of cholecystitis found to occur in 26,499 hospital admissions.[11] Clinical reports appear to indicate clearly that patients who have anginal pain may be relieved after removal of a diseased gall bladder.[12,13,14,15] In two other patients, pain simulating angina reproduced by distention of the common duct through a T-tube was relieved by plastic repair of the common duct in one and after cholecystectomy in another. Thus, it appears that angina may be accentuated by cholecystitis and that distention of, or infection in, the biliary tract may cause pain resembling angina.

There are also reports that electrocardiographic changes indicating myocardial ischemia which are present in patients with cholecystitis may return to normal after cholecystectomy.[15,16,17,18,19] Experimentally, it is reported that these changes also occur in patients after distention of the common duct through a T-tube.[20,21] Similar changes may be brought about in animals if damage to the coronary arteries is produced first.[21]

Arrhythmias, including ectopic beats, auricular fibrillation, AV block, and sino-auricular block, have been described in association with cholecystitis with the disappearance of these arrhythmias following cholecystectomy.[3,14,15,22] These and other arrhythmias, including heart block, ventricular tachycardia, and cardiac standstill, have been produced experimentally by distention of the bile ducts. These phenomena are said to be more apt to occur in the presence of jaundice.[23]

The vagus nerve supplies the thoracic-abdominal viscera with afferent and efferent fibers. Physiologically, there is good evidence that vagal reflexes which are initiated in the abdominal viscera, including the esophagus, stomach, or gall bladder, may produce arrhythmias, cardiac standstill, heart block, and decrease in coronary blood flow,[25,26,27,28,29,30,31] and there is one experimental report which suggests that continuous vagal stimulation will result in my-

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ocardial damage.[32] This makes one wonder if continuous irritation in the gall bladder could produce myocardial damage by constant vagal stimulation.

Anatomically, pain fibers from the gall bladder and heart may overlap.[34] Usually, however, the innervation of the gall bladder is through the visceral afferent fibers from the second to the tenth thoracic segments and possibly the first through the twelfth on the right. In some instances similar fibers run from the fifth to the tenth segments on the left. The pain fibers which originate in the heart and coronary arteries may be transmitted therefrom to the spinal cord at the level of the first through the fifth thoracic segments. Accessory fibers, however, may arise in the gall bladder, which transmit impulses to segments usually receiving such impulses from the heart, and in like manner impulses arising in the heart may be transmitted to the segments usually receiving gall bladder impulses. Thus, pain resembling that of gall bladder disease may arise in the heart and cholecystitis may symptomatically resemble coronary insufficiency.

The following case reports will serve to illustrate examples of the close association between heart disease and the gall bladder:

Case I. Attacks of Paroxysmal Auricular Tachycardia Which Ceased After Cholecystectomy.—Mrs. J. H., a 59 year old lady, was admitted 28 February 1952, and discharged 23 March 1952. She had episodes of paroxysmal auricular tachycardia for the previous ten months not prevented by medications. No clinical evidence of organic heart disease was found. Rheumatoid arthritis and diabetes mellitus were present. Gall stones were found on x-ray and because of the probable precipitation of the attacks of tachycardia through a trigger mechanism by the gall bladder, cholecystectomy was performed. No further attacks have occurred.

Case II. Paroxysmal Auricular Fibrillation.—Mrs. C., a 66 year old lady, was admitted on 4 April 1945, and discharged on 19 April 1945. She had palpitation due to frequent ectopic beats and episodes of paroxysmal auricular fibrillation not controlled by quinidine or digitalis. Anginal pain occurred with the auricular fibrillation but no other evidence of organic heart disease was found. Blood pressure was 130/85. Cholelithiasis was found by x-ray and cholecystectomy was performed on 8 April, 1945. No further attacks of auricular fibrillation have occurred.

Case III. Patient with EKG Abnormalities Which Were Exaggerated Post-Operatively but Returned to Normal Later.—Mrs. M. G., a 53 year old lady, was admitted to the hospital on 1 November 1948. She had four attacks of epigastric cramping pain radiating to the precordium. Nausea, vomiting, and palpitation were associated. No jaundice had occurred.

Physical examination revealed normal heart, blood pressure, and chest. Tenderness was found in the right upper quadrant of the abdomen. Icterus index was 12. Hemogram was normal. EKG taken before cholecystectomy showed T-wave changes indicative of myocardial ischemia.

Cholecystectomy was performed on 4 November 1948. An EKG taken on 6 November 1948, showed further changes, but in one taken 10 November 1948, the T-waves had become upright. The last EKG taken three years later was entirely normal.

She has had no symptoms since referable to the heart or to the gall bladder.

Case IV. Chest Pain Simulating Angina Pectoris Relieved Following Cholecystectomy.—Mrs. H. K., a 29 year old obese lady, was admitted 17 July 1950, and discharged 31 July 1950. She gave a history of pain in the left anterior chest radiating to the left arm of four years duration. The pain was precipitated by exertion and often relieved by rest or nitroglycerine. It was more apt to occur after meals. Bradycardia was noted during the attacks. No jaundice had been present.

Physical examination revealed obesity, normal blood pressure, and normal heart. EKG was normal. Cholelithiasis was found on x-ray.

Cholecystectomy was performed on 24 July 1950. Convalescence was uneventful and no pain has been present since.

Case V. Patient with Coronary Disease and Gall Bladder Disease. Pain Relieved after Cholecystectomy.—Mr. H. M. S., a 72 year old white male, was admitted 14 April 1950, and discharged 7 May 1950. He had an anteroseptal infarction in December, 1947, and during the next four years had chest pain suggestive of angina. Following a severe such attack, jaundice occurred. The gall bladder x-ray showed no excretion of the dye.

Cholecystectomy was performed on 15 April 1950; chronic cholecystitis and cholelithiasis were found. Convalescence was complicated by prostatic obstruction, requiring retropubic prostatectomy on 26 April

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1950. Further convalescence was uneventful and he has had no anginal pain since.

Case VI. Patient with Myocardial Infarction with Symptoms Simulating Gall Bladder Disease.—Mr. S. C. H., a 54 year old white male, was admitted 30 October 1952, and discharged 28 November 1952. He had gaseous eructations after meals for three to four years, and on the morning of admission developed epigastric pain radiating to the right upper quadrant and to the substernal area.

Physical examination revealed epigastric tenderness and tenderness in both upper quadrants of the abdomen. The heart and blood pressure were normal. An EKG was normal. WBC was normal on admission and 12,000 two days later. Gall bladder x-ray was normal. Three days after admission changes developed in the EKG indicative of myocardial infarction. He was given anticoagulants and made an uneventful convalescence.

It is known that there is an increased hazard to the patient with coronary artery disease when he undergoes surgery,[34] and before advising a patient to have cholecystectomy one must consider carefully the risk involved and weigh this against the probable benefits to be derived from the procedure. Hannigan, *et al.*, have reported the mortality following major surgery in 58 patients who had myocardial infarction in the past to be 5.3%.[35] Other reports indicate a mortality of 6.8%[36] to 14.3%[37] in patients with angina pectoris undergoing surgery. Morrison[37] reports an 8.3% mortality of 24 patients with arteriosclerotic heart disease undergoing biliary tract surgery as compared with a general mortality of 2.6% of 1,931 patients.

We have recently reviewed the records of 107 men and 100 women who had cholecystectomy. Of the men, 10 (or 9.3%) died postoperatively. None of the women died. There were 38 men with heart disease, three of whom died as a result of this, and three died as a result of non-cardiac conditions. One must, therefore, consider that a patient with coronary disease, and particularly if that patient is a man, incurs a mortality risk of between 5% and 10% when undergoing cholecystectomy.

It is difficult to determine in a given patient what effect a diseased gall bladder has on the heart, but it would appear that in many patients with symptoms and signs of cholecystitis associated with those of coronary disase or of arrhythmia, the risk of cholecystectomy is justified because of the likelihood that the gall bladder is a trigger mechanism which potentiates the symptoms of coronary disease or of arrhythmia.

In summary:

1. A brief review of the literature indicates that an association between gall bladder disease and heart disease is present.

2. Evidence is cited in support of the view that the gall bladder may initiate afferent reflexes which may bring about electrocardiographic changes, arrhythmias, and chest pain resembling angina.

3. It was pointed out that the differential diagnosis between gall bladder disease and coronary disease may at times be difficult, because of overlapping pain impulses in the spinal cord.

4. Examples are given illustrating the association between the gall bladder and the heart.

5. The mortality of cholecystectomy in patients with heart disease is discussed.

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FAT EMBOLISM*

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The clinical entity diagnosed as fat embolism is still a subject of controversy. The pathological physiology involved has not been clearly determined and there may be some doubt that the condition as such exists. In most cases, the seriousness of the initial illness is far overshadowed by the alarming events that develop when the patient's course seems satisfactory. Most of the earlier work between 1820 and 1880 was done by German investigators. These included Magendie, Virchow, Weber, and Scriba. Warthin[1] in 1913 wrote the classic monograph on fat embolism and little has been added since that time.

In the earlier work it was thought that fat embolism occurred only after trauma to bone or adipose tissue, and typically after fracture of the femur or tibia. However, this phenomenon has been reported in a variety of conditions: all types of bone injury; surgical operations; acute periosteitis and acute osteomyelitis; injuries involving crushing, inflammation, or necrosis of adipose tissue; rupture of the liver; childbirth; eclampsia; tetanus; delirium tremens; convulsive disorders; extensive burns; lipemia due to poisons such as KC104, P, CO; lipemia due to diabetes, pancreatitis, nephrosis, alcohol, and acute febrile diseases; after ether, chloroform and cyclopropane anesthesia; and after I.V. ether administered therapeutically.

The following two patients have been seen in the past year and illustrate the important features of fat embolism.

Case 1. This twenty-four year old construction worker, P. H., received a transverse fracture of the left tibia and fibula when the side of a ditch in which he was working caved in. He received no other injuries. He was transferred by automobile to a hospital in a nearby town where a cast was applied to the leg shortly after admission. He was bright and cheerful when his family visited him that night, and ate the usual hospital evening meal. When last checked by the floor nurse at 11 P.M., his condition was satisfactory. He received morphine for pain on one occasion during the early part of the night, but nothing after that.

At 7 A.M. the next day, the floor nurse found him in a comatose condition, and it was noted that he had twitching of his lips and rigidity of the upper extremities and left leg. His blood pressure at that time was 104/64. During the next two hours, his blood pressure dropped to 90/70, and his condition became worse. He was transferred to a hospital here in Roanoke, and on admission to the hospital, he was deeply comatose, cyanotic, and in severe respiratory distress. He appeared to be in shock, but his blood pressure was not at shock level. Within the next three hours, his temperature became elevated to 103 degrees, and his blood pressure was checked at 104/64. Neurological examination revealed hyperactive reflexes of both upper extremities, and the right lower extremity, but because of the cast, the reflexes in the left lower extremity could not be tested. There was absent abdominal cremasteric reflexes. There was rolling of the eyes from side to side. There were no cranial nerve findings, and no pathological reflexes. Skull x-rays taken shortly after admission showed no abnormal findings. Lumbar puncture revealed clear fluid with a pressure of 180 mm. of water and normal cell count and protein.

Laboratory studies were as follows: Hemoglobin 17 grams; RBC 5,960,000; WBC 22,600 with a shift to the left; hematocrit 45; urine showed 2 plus albumin. The blood urea, blood sugar, and CO2 were within normal limits. A few fat particles were found in the urine but none in the sputum or plasma.

Shortly after admission, his condition became critical, and in spite of normal spinal fluid pressure, the possibility of an intracranial hemorrhage was considered. He was taken to the operating room and bilateral frontal burr openings were made, and a ventriculogram done which revealed no abnormalities in the ventricular system. The pulmonary secretions became thick and tenacious, necessitating a tracheotomy. On the morning after admission, he

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was still comatose and was found to have a fine petechial rash over his abdomen and shoulders. During the next several weeks, the patient's course was very stormy and his temperature ranged from 103 to 104 degrees. He was treated with antibiotics, oxygen, and supportive therapy. During the third week, he began to respond, and gradually improved so that at the end of two months he was able to feed himself and say a few words, although he had considerable mental impairment. Subsequent examinations have revealed that he is slowly but progressively improving mentally.

Case 2. This twenty year old white male, H. H., received a compound comminuted fracture of the right femur in an automobile accident about midnight and was carried to a nearby hospital. He was put in temporary traction, and when his mother left him at 1:30 A.M., he was laughing and talking and had no complaints except slight pain in his leg. It was learned that after eating his evening meal, he had gone riding with friends, and had been drinking heavily at the time of the accident. On the morning after admission, he was found unconscious in bed and was referred to a hospital in Roanoke.

On arrival, the patient was semi-comatose and responded only to painful stimuli. He was in respiratory distress, and there were coarse bubbling rales throughout both lungs. His blood pressure was 116/88. His temperature was 102.6. There was a small abrasion in the right temporal region, and definite left facial weakness, as well as weakness and increased reflexes in the left arm, and slightly increased reflexes in the left leg. There were no pupillary changes.

On admission, his laboratory studies were as follows: Hemoglobin 13.5 gms.; RBC 4,500,000; WBC 14,300 with a normal differential; total eosinophil count was 0. His catherized urine showed a 3 plus albumin. No fat particles could be found in the plasma, sputum, or urine.

It was felt that this patient might possibly have an intracranial hemorrhage, and bilateral burr openings were made, but no evidence of a hemorrhage was found, and his ventricular fluid was clear. The brain was not tense and no ventriculograms were made. Because of extreme respiratory distress, a tracheotomy was done after the burr holes were made.

Shortly after the patient was returned from the operating room, his blood pressure dropped to 98/64, but gradually rose to normal level over a period of twenty-five minutes. On the morning following operation, he was found to have a fine petechial rash over his chest and abdomen with a few scattered spots on his extremities. See Fig. 1. His respirations had improved, although he remained comatose. Approximately a week after he was admitted to the hospital, his mental symptoms began to clear, and his temperature, which had been elevated during the previous week, began gradually to return to normal. On the seventeenth hospital day, he was taken to the operating room and an intramedullary bar inserted into the right femur without complications. He was discharged from the hospital on 5/31/53, and was alert but mentally sluggish. He has been seen for follow-up visits since his discharge, and as far as can be determined, has returned to his normal mentality and has no neurological sequelae.

INCIDENCE

In routine autopsies some degree of fat embolism has been found to occur frequently in cases of traumatic injury, especially with bone fracture[2]. However, the mortality due directly to fat embolism is small and unpredictable. Males predominate in the ratio of 8:1 and then most often in the fourth decade. It is rarely seen in children. Alcoholics are more subject to fat embolism than non-alcoholics. Warthin found fatty embolism responsible for about 2% of deaths in 560 routine autopsies. Most authors agree that traumatic fat embolism occurs more commonly than is generally known and many fatal cases have been diagnosed as shock, coma, apoplexy, alcoholism, brain hemorrhage, pulmonary embolus, and coronary thrombosis.

CLINICAL PICTURE OF FAT EMBOLISM

The diagnosis is dependent on the awareness of its possible existence in a given patient. The most important feature of traumatic fat embolism is the "free interval" following injury during which the patient's condition is satisfactory. This latent period is usually a few hours, but has varied from 30 minutes to 9 days[1].

Without warning, pulmonary and/or cerebral symptoms appear and the patient's condition quickly becomes critical. Pulmonary symptoms consist of dyspnea, cough, cyanosis, frothy or bloody sputum. Pulmonary edema and bronchopneumonia are frequent, and cor pulmonale may develop. Cerebral symptoms include insomnia, irritability, disorientation, coma, convulsions, rigidity, and occasionally lo-

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calizing cranial nerve signs. Cerebral damage may last for weeks and may be permanent.

Elevation of temperature occurs promptly and may be extreme. This usually subsides after several days. Within 24-48 hours a fine petechial or even hemorrhagic rash may be found chiefly on the abdomen, chest, and shoulders if carefully searched for. This rash is easily overlooked and usually disappears in 2-3 days. The fundi may show emboli

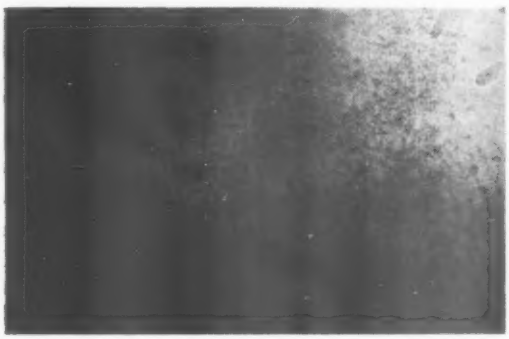

Fig. 1. Petechial Skin Rash, Case 2.

in the retinal vessels[3], and hemorrhages and exudates may be seen at times. It is characteristic in traumatic cases that the degree of embolism is far greater than one would expect from the extent of the injury[1].

Both of the cases presented were admitted to the hospital in a comatose state with fever. There were signs of marked pulmonary embarrassment with large amounts of frothy secretions necessitating tracheotomy. The respiratory distress was markedly relieved by repeated adequate suctioning through the tracheotomy tube. Both patients had findings which suggested possible intracranial hemorrhage. Spinal fluid examination and burr hole explorations were done in each case and proved negative. In addition, on the morning after admission a fine petechial rash was observed on the abdomen, chest and shoulders. The first patient developed mild shock several hours after the onset of coma, and the second had only a transient drop in blood pressure at the time of operation. The "free interval" in *Case 1* was about 12-15 hours, and about 4-5 hours in *Case 2*. The latter patient had been drinking heavily at the time of the accident. The first patient ate the usual hospital evening meal, and the second had eaten a meal high in fat about 5-6 hours before the accident. Both were healthy, vigorous young men without history of previous serious illness.

Laboratory Findings

A survey of the literature as to laboratory tests diagnostic of this condition makes it evident that there is at present no satisfactory definitive test for fat embolism. The diagnosis must be made primarily from the clinical picture and in fatal cases confirmed by post mortem examination.

When present, fat in the sputum and urine are helpful suggestive findings. However, Du Toit[2] and others point out that fat appears in the sputum late and may be positive in other diseases. Warren[4] feels that lipuria is not of much value and seldom develops before the fourth day. The urine must be obtained by catheter without the use of lubricants and as the fat floats to the top of the bladder, care must be taken to empty it completely. Microscopic examination and the "sizzle" test are usually satisfactory.

Blood fat estimation and examination of the serum for fat have been of little value. An explanation for this could be that by the time fat embolism is suspected in most cases the fat particles have been strained out and the damage done. It is also conceivable that the fat may go back in solution easily. In neither of the cases presented in this paper was the serum cloudy. However, considerable time had elapsed before the examination was made. A few fat particles were found in the urine of the first case.

Many authors emphasize the importance of unexplained anemia in suspicious cases. This is not a constant finding and is probably due to blood loss into the tissues or to hemolysis. Schneider[3] feels that fat droplets in the blood have little value *per se* as the size of the globules is of much more importance than the total number or the quantity of blood fat.

Whitson[5] states there is no pathognomonic laboratory finding in fat embolism. The spinal fluid is usually clear and chest x-rays may show densities, suggesting bronchopneumonia.

Differential Diagnosis

Widespread bronchopneumonia, pulmonary edema due to intrinsic heart disease, and pulmonary embolus from blood clot formation may simulate the findings of fat embolism and at times cannot be completely excluded until the entire clinical picture of the condition has developed. Septicemia due to any

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cause and bacterial endocarditis can usually be ruled out by proper laboratory study and clinical observation. Midbrain injury, concussion, and epidural or subdural clot have to be considered in all cases, especially following trauma. If coma and localizing neurological signs develop, burr openings and ventriculography may be necessary to rule out a surgically correctible lesion.

Undetected diabetes, poisoning, hypoglycemia, and uremia can cause coma but are easily diagnosed when thought of. Delirium tremens is always a possibility in chronic alcoholics but it should be remembered that fat embolism occurs more frequently after the intake of alcohol.

If unexplained neurological and pulmonary symptoms develop at child birth or during or immediately following operation, the possibility that fatty emboli are responsible should be considered. It is logical to assume that fat embolism is more likely to occur in patients with lipemia due to any cause.

Delayed shock, if progressive and fatal, can cause almost all of the findings seen in fat embolism[5]. However, as borne out by the above two cases and by numerous other reports, shock is not always present and, when it does occur, is a late finding.

PATHOLOGICAL PICTURE

According to Warthin[1], Du Toit[2], Warren[4] and others, *the lungs and the brain are the sites of the primary damage in fat embolism.* Injury to other organs can occur but rarely causes symptoms. The lungs are heavy and large and the cut surface shows a marbled appearance with zones of hemorrhage alternating with emphysema. A frothy blood stained exudate is present and may appear fatty on close examination. Bronchopneumonia is found after the first 48 hours. *On microscopy fat globules can be seen blocking the capillaries* and at times appear in the alveoli. The lung structure shows congestion, edema and emphysema. In severe cases the pulmonary artery may be almost completely filled with fat particles. If the pulmonary involvement is extensive, the right side of the heart is usually dilated. Occasionally actual bits of bone marrow have been found in the pulmonary artery.

The brain shows hyperemia of the meninges and cortical vessels. The gray matter may show little involvement, while the white matter will show petechial hemorrhages. Areas of softening may occur, particularly in the brain stem. The capillaries contain fat emboli and the choroid plexus frequently shows widespread damage. There is perivascular myelin degeneration and usually little neurological reaction.

Fatty emboli may be found in any of the capillaries of the body and there may be congestion as seen in the kidney or actual petechial hemorrhage as seen in the skin. There may be an increase in liquid fat at the site of the injury.

In the pathological technique used it is important that fresh material be stained with sudan III, osmic acid or other fat stain after frozen section. The material is mounted in glycerine-gelatin or formol.[1]

PROGNOSIS

There is no unanimity of opinion as to the prognosis in a given case. Death is rare after the first four days. *The cerebral damage, if severe, may take months for recovery and even then may not be complete.*

TREATMENT

There has been no satisfactory treatment of fat embolism once it has occurred. The immediate use of oxygen and measures to prevent the development of shock can be expected to reduce the amount of damage to the lungs and brain. If pulmonary secretions cannot be easily controlled by the usual oral or nasal route of suctioning, then tracheotomy may be life saving. Antibiotics should be begun at once to prevent the development of serious pneumonia. Good nursing care is imperative.

DISCUSSION

It is apparent from a review of the literature that the basic cause of fat embolism has not been definitely established and needs further investigation. The theory proposed is that fat emboli gain entrance to the venous system at the site of injury of bone marrow or adipose tissue. The fat particles are washed through the hematoma into the ends of veins opened by trauma and thence pass to the lungs. Some of the fat is squeezed through the lung capillaries and passes to the arterial circulation. Warthin,[1] in his classic review of the subject, had little doubt that the source of the fat was the injured tissue and most authors still accept this view. Several serious inconsistencies have been observed, however, in the cases reported through the years. The most important of these is the occurrence of the condition in other diseases. Then, too, some patients have only cerebral symptoms and, as Whitson[5] points out, it

is difficult to see why emboli passing through one capillary bed will cause blockage in another. Warthin[1] first noted that the total amount of fat liberated may at times be unbelievable, and may be far out of proportion to the liquid fat known to be contained in an injured long bone. The femur in man has been shown to contain approximately 65 cc. of liquid fat, and Lehman and Moore[6] have demonstrated that even in dogs 100-120 cc. of liquid fat must be injected rapidly in order to produce a lethal outcome using cottonseed oil. It is illogical to assume that the entire amount of fat in a traumatized bone in man can drain into the venous system and that even if this did occur that the process could be rapid enough to overwhelm the body defense mechanisms.

The small bits of marrow occasionally found in the pulmonary artery and its branches after bone trauma are insufficient to cause serious symptoms and represent an incidental finding for the most part. These undoubtedly break off and enter the venous channels in the bone before thrombosis occurs.

As Wrist and Johnson[7] have written, a careful study of healing fracture areas and other wounds fails to demonstrate fat embolism taking place. On the contrary, Whitson[5] feels that with injury, tissue tension collapses the veins and thrombosis quickly occurs. Even the vessels in bone channels show thrombosis shortly after injury, although the scant areolar tissue collapses at a slower rate. Some liquid fat could enter the venous system at this time, however, this portal of entrance is sealed off long before the "free interval" seen in most cases of traumatic fat embolism has passed. The idea that negative pressure in the veins "sucks" fat from the bone marrow is untenable. Du Toit[2] thinks there is no real evidence that the lymphatics play a significant role as portals of entry of the fat.

Lehman[6] and others have stated that blood chemistry alteration is important in the development of fat embolism; however, Scuderi[8] concludes that there is no convincing evidence that the composition of blood including fat is significantly altered following fracture.

Whitson[5] states that a reduction in the oxygen carrying capacity of the blood is the primary factor in the production of fatty degeneration and other changes ascribed to fat embolism. He points out that anoxemia and shock will produce identical changes in the tissues as does embolism. He does not account for the fact that in most cases shock, if present, occurs late in the illness. He concludes that intravascular fat is the result rather than the cause of capillary stagnation. This is an interesting viewpoint, but does not fit the facts any better than most other theories about fat embolism.

As seen in the cases here reported, the signs and symptoms of fat embolism were present several hours before there was any evidence of shock, and even then was not severe in either case.

The extreme changes described by Whitson[5] and others are seen in terminal shock, and it is not unreasonable to expect the tissue changes to be similar, as anoxemia is present in both, but from a different cause. There is certainly no real evidence that impending, undiagnosed or mild shock can produce all the changes seen in fat embolism. If Whitson[5] were correct, it is difficult to see why the clinical picture of fat embolism is not present in all cases of severe shock. Prolonged anoxemia and shock due to any cause can result in the "vegetable" patient, but the clinical picture is not that of fat embolism. Then, too, many careful and reliable observers have reported massive amounts of fat in the vessels and this cannot be explained by fatty degeneration. There can be no doubt that fat embolism has been produced experimentally by the injection of liquid fat into animals even though the way embolism occurs clinically is still not clear.

If the fat does not come from injured bone marrow or adipose tissue, then it must come in some way from the blood itself. There are now numerous papers in the literature relating to the behavior of lipid material in the blood which are pertinent to the problem. Lehman[6], Du Toit[2], and others have considered the possibility that the blood might serve as a source of the emboli, but reached no definite conclusion about the matter. Lichenstein[9] also has speculated that emulsified fat in the sera or possibly the lipid in the red cell envelope might be the origin of the fat. Lehman and Moore[6] have already suggested that this phenomenon may be due to fat normally in the blood aggregating into larger particles as a result of certain physical and chemical changes in the blood. As already noted, however, there are no consistent changes and there is no correlation to blood volume, hematocrit, anemia, etc.[10]

Normally, about three-fourths of the fat in the blood is in solution and is attached the beta lipoprotein[11]. Physiologic hyperlipemia is seen after the ingestion of fats with a peak in the fat tolerance

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curve occurring about 3 hours after the ingestion of the meal. The turbidity seen in hyperlipemia is due primarily to an increase in neutral fat. When the cholesterol or phospholipides are elevated, the serum is clear. In essential hyperlipemia, anoxia, nephrosis, poisoning due to CHC14, chloroform, etc., an increased turbidity in the plasma has been observed[12]. Ahrens and Kunkle[13] point out the importance of the stabilizing effect of the phospholipids on the serum lipids. In the condition of essential hyperlipemia, Joyner[12] and others have found the phospholipids to be low. Boyd[14] has concluded that the phospholipids tend to maintain the other fat fractions in a supersaturated state in the serum and, when the phospholipid content of the serum diminishes, the lipids come out of solution as chylomicrons, forming a milky emulsion. According to Holden[15] the normal serum neutral fat is 0-150 mgm.%. Visible cloudiness usually occurs at about 300 mgm.%. The initial rise in neutral fat is detectable about 1 hour after eating and returns to normal levels in 4-6 hours.

Harman and Ragaz[10] have published a careful study on the pathogenesis of fat embolism. These authors point out that the response of the host in fat embolism is variable and that the condition of the patient or experimental animal is as important as the action of the fat on the host. They found that fat particles above 8 microns in diameter may cause embolism, whereas smaller particles cause no difficulty. Homologous liquid fat was injected into rabbits and it was found that approximately 1.0 cc./kg. would cause 100% mortality in the animals. In another experiment these investigators found if the animals were traumatized 7 hours prior to the injection that 0.45 cc./kg. of fat caused the mortality to increase from 16.6% to 50%. Dehydration and hemoconcentration had no significant effect on the experiment. They found, if oxygen was administered, that the 50% mortality dropped to zero, although some tissue changes could not be prevented. The initial result depended mainly on the quantity of fat injected, whereas the subsequent course of the animals depended on the action of the fat upon the tissues. Fat does not affect the tissues in an indiscriminate way such as would be the case if there was a generalized chemical reaction. This action must be decided by factors independent of the nature of the fat itself.

Scuderi[8] and others have shown that hydrolyzed fat is much more toxic than neutral fat from which it is derived. It produces an intense exudate in the lungs of the experimental animal. Warren[4], however, was able to demonstrate neutral fat in a few of his 100 cases. He found that oleic acid is 7 times as lethal as neutral bone marrow fat. It was suggested that the pathologic changes in fat embolism are probably in part due to the action of free fatty acids and soaps liberated by the action of tissue lipase as well as to mechanical plugging. Gomori[16] and others have demonstrated that lung tissue is high in this enzyme. This may well be a factor in cases of associated pancreatitis with fat embolism.

Harman and Ragaz[10] further show that no morphologic alteration in lung tissue is found when massive pulmonary vessel plugging occurs following paraffin injection where no fatty acids are formed.

Robinson[17] feels that fat is removed from the circulation by four mechanisms: excretion through the kidneys, excretion through the liver, reticuloendothelial system, and blood and tissue lipase. The real problem, however, is how the fat leaves the blood stream. Interesting experimental work along this line has recently been reported by Swank[18]. He found that normally there is a physiological clustering of fat chylomicrons in the blood after a fatty meal. Maximum clustering occurred at about 8-10 hours and was greater with larger amounts of oral fat. These larger fat particles are then presumably strained out of the circulating blood by capillaries and reticuloendothelial cells. He feels that this suggests a possible mechanism for multiple capillary embolization and, if sufficiently numerous in any one area, that disseminated lesions, both functional and pathological, could result. It was further observed that the tendency to cluster varied in different individuals. It was noted that a high fat diet and pregnancy increased the tendency for clustering. This is interesting in view of the cases of fat embolism that have been reported in pregnancy. I.V. heparin has been found to promote clustering[19] as well as histamine.

Marder *et al*[20] recently have shown that by chylomicron counts and nephelometric determinations on serum examined after ingestion of a fatty meal there is slower absorption of fat in the aged than in young people. Ingle[21] has shown that adrenal cortical hormones promote the absorption of fat from the intestines.

The role of fat solvents in fat embolism must be

considered. It is known that *alcoholics are more prone to fat embolism than non-alcoholics* and also this condition has occurred following i.v. ether therapeutically as well as following ether and cyclopropane anesthesis. Du Toit[2] suggests that the fat emulsion in the serum is less stable in alcoholics and postulates that this is due to frequent dilution with alcohol which is a fat solvent. In the case of fatal fat embolism following i.v. ether administration[9] the patient was allowed to eat his noon meal about 2 hours before the ether drip was started. It seems probable that fat solvents may in some way make the fat unstable; however, a better explanation for the role of fat solvents in fat embolism would be that more fat remains in solution than normally as the physiological removal by clustering might be delayed until the effects of the solvent have worn off. At this point, massive clustering could occur and initiate serious embolism.

The possible role of ingested food in fat embolism has been neglected and may well be of extreme importance. In *Case 1* here reported the patient was allowed to eat his evening meal and it was several hours after this that embolism occurred. *Case 2* ate his evening meal and then drank before the accident which could favor the development of fatty emboli. In general, it may be stated that the majority of diseases in which fat embolism has been reported are conditions in which the patient would be most likely allowed to eat a fairly normal diet. This is particularly true in fractures. In most other surgical procedures, the patient is fasted some time before and after operation.

There would still appear to be some other factor which is primarily involved in the development of this phenomena. *When one considers the multiplicity of conditions and diseases during which fat embolism has occurred, the most striking thing that is common to all is pituitary adreno-cortical stimulation. In many cases, particularly following trauma, the "free interval" corresponds perfectly with maximal response of the adrenal cortex to ACTH.* Rich *et al*[22] have recently reported marked lipemia in dogs treated with cortisone. It has been recognized for some time that the adrenal cortext exerts an effect on fat metabolism, although the exact nature of the effect is not known. Price and Bloom[23] have shown that, in rabbits, prolonged cortisone treatment causes large quantities of serum lipoproteins of the SF 80-400 class to appear. This was converted serially to the lower SF values when the cortisone was stopped. Aldersberg[24], in studying hormonal control of the serum lipid partition in man, observed incidentally that the fasting sera of many of his experimental subjects given ACTH and cortisone was turbid or opaque due to lipemia. He speculated that this may indicate the production of less soluble lipid or a lipid of much larger particles than normal, or possibly the conjugation of the lipoprotein in an abnormal manner. He feels that the hormonal changes produced by cortisone and ACTH can easily upset the ratio between free and conjugated lipids, especially since a change in the globulin fractions has been found by electrophoretic determination.

The phospholipids have not been measured in cases of fat embolism, however, Rhon[23] reports a case of severe hyperlipemia in a pregnant woman who at delivery developed acute pancreatitis and in whom the phospholipids were low. In view of the relation of the phospholipids to the stability of the fat emulsion previously referred to, it would be interesting to do this determination in cases of fat embolism. It would also be interesting to study the effects of massive amounts of ACTH on the serum phospholipids at the time of maximal adrenal stimulation. Adlersberg's[24] results were inconclusive on this point, but his procedures were not planned with this in mind.

The presence of alimentary lipemia at the peak of hormonal effects on the plasma lipids might well be the trigger mechanism for the production of fat embolism. In addition, there are probably other unknown factors such as individual susceptibility and the plasma phospholipids which influence the stability of the fat emulsion.

PROPHYLAXIS

Everything possible should be done to prevent further injury in the handling of severely traumatized patients. If the possible role of alimentary lipemia in fat embolism can be established, it would seem logical to eliminate fat from the diet of patients during acute stress of any type in the first few days; this applies particularly to bone fracture. Fat solvent anesthetics should be used with caution in such patients.

SUMMARY

1. Fat embolism does exist as a clinical entity and still has to be diagnosed by the clinical picture.

2. Fat embolism occurs in a variety of conditions

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but is seen typically after trauma, especially fracture of bone.

3. There is now considerable evidence that this condition in some way results from lipemia or dissociation of the plasma lipids with multiple capillary embolization, and is not due to liquid fat in the marrow draining into the venous system.

4. Several possible mechanisms operating in the production of fat embolism are suggested:

a. Alimentary hyperlipemia.

b. Normal physiological clustering of chylomicrons.

c. Instability of fat emulsion in the plasma due to lowering of phospholipids or other unknown factors.

d. Fat solvents, such as ether and alcohol, which enable the plasma to become supersaturated with fats.

e. Hyperlipemia resulting from adrenal cortical stimulation by ACTH.

5. Individual susceptibility for the occurrence of this condition probably varies considerably.

6. Treatment is supportive. Oxygen therapy and prevention of shock are of extreme importance.

7. There is need for further basic research on this problem.

ADDENDUM

Dr. J. C. Forbes at the Medical College of Virginia has called our attention to a recent article by Fullerton (British Medical Journal: Aug. 1, 1953) concerning the relationship of alimentary lipemia to blood coagulability. This may also be an important factor in the production of fat embolism.

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THE PSYCHOGENIC ETIOLOGY OF PEPTIC ULCER*

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The title of this paper is not meant to imply that psychogenic factors are the sole cause of peptic ulcer or that all peptic ulcers are of psychogenic origin. It means that in this paper we will be discussing the role of these factors in the causation of most cases of ulcer and the psychodynamics and mechanisms by which this occurs according to widely held theories.

In psychosomatic medicine, which simply means good medicine, it is a basic premise that emotions affect the physiological functions of the body in very definite and tangible ways. This has been so thoroughly documented through the experiments and observations of many workers from Beaumont through Pavlov, Cannon and Wolff, and the personal and professional experiences of everyone in this conference as to leave no further room for argument. It is also generally agreed that disturbed intensified emotions lead to disturbance of function of many organs and systems of the body, particularly those under controls of the autonomic nervous system, and that prolonged or frequent disturbance of function leads as a rule to structural alteration of the tissues involved. We know something of the neurological and hormonal pathways through which this is effected and something of the altered physiology and pathology which results. Undoubtedly, there is more to learn all along the line.

Returning specifically to the natural history of peptic ulcer, we would outline its development as follows: Disturbed interpersonal relationships more or less consciously perceived in the cortex, transmitted through the thalamus to the hypo-thalamus and pituitary, then into the autonomic pathways, particularly, and more or less selectively in this case, to the vagus nerve, activation of which leads to hypermotility, hyperemia and hypersecretion of the stomach—a condition apparently conducive (if sufficiently prolonged, intense or frequently recurring), to ulceration of the mucosa. Certain other x-factors may also have to be present at the site of ulceration, such as an excess of lysozyme, in order to produce actual ulcer formation. On this much we can all probably agree, so we must push on to something more controversial.

*Presented at the Conference on Gastro-Enterology, November 20, 1953, at Charlottesville, Virginia.

In medical school text-books, it has long been customary to say that the typical ulcer patient had an asthenic body build, was an energetic, driving, ambitious, go-getter, and that the incidence was much higher in men than women, and during the third and fourth decades of life. The relevance of these clinical observations to the cause of ulcer was not clear, but thought to have something to do with nervous tense individuals being subject to dyspepsia, nervous indigestion, excessive acid output, hypermotility of the stomach and poor dietary habits, and that men from 20 to 40 worked harder and worried more. The empierical common-sense treatment was to encourage these individuals to slow down, take it easy, stop worrying, stop smoking or drinking, go on a diet of milk and cream at frequent intervals and preferably come into a hospital for rest and nursing care. This usually worked remarkably well, and still does, at least, temporarily. But some people are not content to let well enough alone and want to know WHY—which explains, I suppose, why a psychiatrist is on this panel—because the psychiatrists think they have come up with some answers into which everything fits very neatly.

It is also generally recognized that the real problem of ulcers is not the cure of the acute ulceration. It will heal under a good medical regime, or it can be very satisfactorily eliminated surgically. The real problem is the recurrence, or one might put it that the real problem is with the person who gets ulcers—and that is where psychiatry presumably comes in.

Two other clinical observations are pertinent before proceeding with speculation on what we like to call the dynamics. It has been noted that many ulcer patients experience a nocturnal rise in hydrochloric acid during sleep, which cannot be accounted for simply by absence of food or anti-acid intake. How does this happen during sleep when the person is at rest from the work, worry, etc., that are supposed to stimulate secretions?

It has also been observed that there are a fair number of exceptions to the classical picture of the ulcer case as a business tycoon or even the worrisome, overly conscientious clerk. Some are literally

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bums, ne'er-do-wells, irresponsible, markedly dependent types.

Psychiatrists proceed on the assumption in common with Gilbert and Sullivan that "things are seldom what they seem." It applies here in our theory of the dynamics of the ulcer case. We believe that the typical "ulcer personality," far from being really an aggressive, striving, ruthless, independent man, is actually passive, dependent, anxious, hungry for love, wanting to be taken care of like an infant, but he cannot accept this aspect of himself, reacts strongly against these wishes or needs, and overcompensates in the opposite direction; thus, appearing on the surface to be the type of person usually described. He literally deprives himself, starves himself of the very satisfactions he craves. In the case of the atypical type mentioned above who is overtly passive and dependent, he is usually deprived by external circumstances, unable rather than unwilling to gratify his receptive needs for love and dependency.

To quote from Dr. Alexander, who has, perhaps, contributed most to our modern conception of the psychodynamics of stomach ulcer:

"There are few vegetative functions which play such an important role in emotional life as does the ingestion of food. From early life on, eating is associated with the feeling of security, of receiving love and care, and also with greed, possessiveness and envy. The neurotic conflicts centering around these basic emotions may contribute to such things as disturbance of appetite as in bulimia and anorexia, of swallowing, as in cardiospasm, and nervous vomiting, and to dysfunctions of the digestive tract such as duodenal ulcer, constipation and diarrhea. What seems to be best established is that the accentuation and inhibition of the wish to receive love and protection being deeply associated from infantile experience on, with feeding, may activate or inhibit almost any phase of the incorporation and digestion of food. Those functions which are under autonomic innervation are activated or inhibited by emotional stimuli on the principle of the conditioned reflex. This is illustrated by the following sequence: the wish to be loved, to receive, to be taken care of, is associated on the psychological level with the wish to be fed and on the physiological level, with preparation for being fed, that is, increased gastric secretion, hyperemia and hypermotility."

Some workers in the field, particularly those without psychoanalytic orientation are inclined to feel that too much stress is laid on the specific personality type and the specific conflict situation—the deprival of passive receptive love. They feel and have some experimental and clinical evidence to indicate that the problem is simply one of anxiety to which some individuals are inclined to react with one organ and some another. In the case of ulcers, the patient is a predominantly stomach reactor. Others experience or express emotions more through the colon or the cardiovascular system, or the respiratory system or the skeletal musculature.

It is perhaps not too hard to visualize that the infant who is pacified by having a nipple, be it breast or bottle, stuck in his mouth no matter what the source of his discomforture may be, becomes conditioned psychologically and physiologically to expect to be fed whenever he is unhappy, and that residuals or equivalents of this conditioned reflex may be carried over into adult life. Other examples of this reaction pattern are: thumb-sucking, smoking, chewing, eating, drinking, when done to relieve anxiety or tension, to enhance an illusion of well-being, safety, comfort and love, reminiscent of infantile bliss at the mother's breast. The physiological processes associated with ingestion have become, in these individuals, associated with any state of discomfort or tension.

Wolff and Wolf at the New York Hospital, working with their subject who had a gastric fistula, noted by direct visual observation that, during states of fear or depression, a predominantly sympathetic stimulation occurred, resulting in hyposecretion, hypomotility, mucosal pallor and decreased mucin production. Emotions of resentment, anger and anxiety were associated with hypersecretion of acid and pepsin, hypermotility, hyperemia and increased mucin output—in other words, parasympathetic activity and also a state of preparedness for receiving food.

But, one may well say, everybody at one time or another experiences such emotions but they don't all get stomach ulcers. There is usually in ulcer patients other evidence of autonomic instability, such as excessive pallor, sweating, bradycardia, tachycardia, urinary frequency, spastic colitis, etc. It is not autonomic nervous system disturbance alone but the particular imbalance which occurs in the conflict situation that appears to favor development of ulceration. A so-called normal person under a similar situation might get only transient heartburn

but the character structure of the ulcer patient is such as to react with sufficient intensity and duration to cause actual ulceration.

The question of nocturnal hypersecretion and pain in ulcer patients, as noted above, has led to some skepticism concerning the psychologic etiology. Undoubtedly, one factor is the absence of buffering food. In addition is the fact that much of the psychologic conflict thought to give rise to physiologic reactions conducive to ulceration is of an unconscious nature and hence goes on waking or sleeping —in fact, during sleep has more complete and unimpeded sway.

The customary medical treatment of ulcer, so often successful in treating the acute phase, fits well into these theories of etiology. The patient who characteristically drives himself, resists giving in to his pain, who does not take care of himself, is frequently ashamed to appear soft, to admit his wish to give up and be taken care of. However, when he finally becomes sick, has a hemorrhage or perforation or is forced in one way or another to admit he is sick, put himself in the care of a physician and enter a hospital, he then has a face-saving, acceptable excuse to let down and accept care, frequent feedings, backrubs, mothering nurses, kindly paternal doctors, etc. This treatment has rationale and efficacy over and above the purely biologic considerations. But when he is recovered, when he can no longer justify to himself this life of indolence and self-indulgence, he has to be up and at 'em, and to hell with doctors, diets and medicines. The other common type, the overtly passive dependent, or the person simply deprived by circumstances of adequate satisfaction of these needs also responds to this regime of T.L.C., but the problem with him may be in getting rid of him. He laps it up and is unashamed.

It is important to stress here that one must not fall into the error of too much generalization or expect typical, classical stereotypes. It is probably true that the basic conflict of the ulcer patient, namely, lack of satisfaction of dependent receptive "oral" needs and resulting anxiety and resentment, is nearly always to be found, but the variations in the individual picture are as infinite as individuals. It is necessary to study each case as an individual, his personality structure, his reaction patterns and the circumstances of his life, his environment and his interpersonal relationships, past, present and future, in order to really understand why he gets ulcers.

To quote Dr. Leon Saul: "What one must learn is to recognize this common feature—the needs and how they are frustrated—in the very different forms in which it presents itself in different cases. The treatment will depend on what one finds."

Pernicious Anemia Treated by Nasal Spray Inhalation.

An effective and inexpensive means of treating pernicious anemia—a serious blood condition—has been found by the administration of vitamin B-12 through inhalation or use of a nasal spray.

Twelve patients who had a return of the disease after its apparent cessation were successfully treated by this method. In 20 other patients the symptoms of the disease were held in abatement for periods up to 18 months with the same treatment.

Drs. Raymond W. Monto and John W. Rebuck, Detroit, wrote in the Archives of Internal Medicine, published by the American Medical Association, that this method of therapy has produced no evidence of toxicity or sensitivity.

Previous methods of treating pernicious anemia, including the eating of sufficient quantities of fresh liver to be effective, oral administration of liver extracts and massive doses of vitamin B-12, and injection of vitamin B-12, have been successful, but have proved to be unpalatable or very costly, the doctors pointed out.

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REHABILITATION FROM THE VIEWPOINT OF A PHYSICIAN IN INDUSTRY*

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Unfortunately, the word rehabilitation brings to mind far too frequently the mental picture of paraplegics and amputees. While this group of individuals is a very important one (and incidentally is a job which is being well done), the number of individuals with such difficulties in the average physician's practice is so small that he, like most of us, is prone to think of rehabilitation as something the other fellow must do. In reality, rehabilitation is the final phase of treatment of every patient. The degree to which it must be applied varies with the nature of the disability.

Failure to consider this phase of your patient's recovery can result in considerable economic loss to the individual and his family through failure to return to his occupation as soon as he might have, commensurate with proper protection of his health.

Early ambulation has shortened the recovery period for most surgical cases and hastened their rehabilitation. Physical medicine would do the same for many fractures which are now kept in casts for weeks and subsequently may require weeks of physiotherapy. Earlier utilization of physical medicine could help considerably.

Industry can play an important part in rehabilitation by placing injured individuals on jobs which will permit safe use of the injured part of body or which will permit gradually increasing activity. We, in industry, have found that such measures hastened recovery and often prevent permanent disability. Gradual increase in activities and proper work placement can aid in physiological recovery.

Such practices require an understanding and willing industry. Unfortunately, we still have with us industries whose attitude is "full work or no work". In other words, let compensation insurance pay the bill until this individual is completely recovered. This policy is not good for the employee. It encourages malingering and the development of traumatic neurosis. Forward looking insurance companies are encouraging physical medicine and rehabilitation centers, both as humane endeavors and an economic procedure. Shortsighted insurance companies (of which there are far too many) adopt the attitude of settling with the individual as soon as possible, leaving him as a public charge for you and me to support through our welfare programs. Such practices should be prevented. Changes in our compensation legislation have been of some help. However, we still are not utilizing our physical medicine and rehabilitation facilities sufficiently. Insofar as compensation cases are concerned, no insurance company should be permitted to close the case until the compensation commission has adequate assurance that the individual has been rehabilitated as much as possible, considering the nature and extent of the injuries.

Large industry has in many instances recognized the value of aiding in the rehabilitation of its employees, but large industry is only a small employer in Virginia. Small industry employers have the majority of the wage-roll workers. You, as physicians, can influence the management of this type of industry to aid in rehabilitation, not only of the injured but the chronically ill—the arrested TB., the rheumatic heart disease, the asthmatic, chronic nephritic, etc. Obviously, you must acquire knowledge of work requirements if you are to intelligently aid in work placement; see the job in actual operation and ask yourself, "Could I do that job safely without harm to my health if I had what my patient has?" This is rehabilitation. It is our responsibility. Are we fulfilling our duties.

We must bear in mind that what is applicable to those injured in industry is equally applicable to those injured elsewhere. When one examines the statistics, we find that industry, although it is growing rapidly, is decreasing its incidence of injuries. In 1952, 34,500 workers were killed off the job and 15,000 on the job. There were 2.6 million off-the-job injuries and 2 million on-the-job injuries reported. It is to be noted that the 49,000 deaths account for a little more than half of the total accidental deaths, which were 96,000. Thus, the oppor-

*Read before the annual meeting of The Medical Society of Virginia at Roanoke, October 18-21, 1953, as a part of the Panel Discussion on Rehabilitation.

tunity to rehabilitate will arise more often from sources other than industry. Are you missing these opportunities?

A few examples will serve a purpose: We have an expert lathe operator who lost an eye. He is completely rehabilitated and is still an expert lathe operator. Many employees with unilateral amblyopia are doing as good work, and at times better, on machines, as those with no impairment of vision. An amputee from peripheral vascular disease (age 53) has an artificial leg and is performing a day's work. Several employees with mixed rheumatoid and osteo-arthritis are carrying on with remarkably good attendance records in view of their handicaps. One case of multiple sclerosis has had an excellent attendance record and has been doing a good day's work in the Machine Shop.

Burt Hanman, in his book, "Physical Capacity and Job Placement," has emphasized the positive approach. He points out that we should place employees with reference to their abilities and not their handicaps. After all, each and every one of us is handicapped in one way or another with reference to various and sundry occupations.

Major Surgery in Elderly Patients Relatively Safe.

Major surgical operations can be carried out in patients over 70 years of age with reasonably low risks. Therefore, adequate surgical care should be made available to older persons not merely to save life but also to relieve discomfort and disability, in the opinion of Drs. John D. Stewart and Guy S. Alfano, Buffalo, N. Y. associated with the department of surgery of the University of Buffalo (N. Y.) Medical School and the E. J. Meyer Memorial Hospital.

They based their conclusions on two studies of persons over the age of 70 years who underwent surgical procedures. One study consisted of 204 general surgical patients who underwent 290 major operations, and the other of 43 patients who had major abdominal surgery.

The average age of the 43 patients was 74.4 years, the oldest being 88, the doctors wrote in the current (February 20) Journal of the American Medical Association. Four of the patients (nine per cent) died; two deaths were caused by heart conditions.

The average age for the series of 204 patients who underwent 290 general surgical procedures was 76.8 years, the oldest patient being 94. The overall mortality rate was 13 per cent (39 patients). However, the doctors pointed out, 21 of the 39 deaths probably had little relation to the surgical operation; 17 of the 39 patients who died had advanced cancer. If these deaths were excluded, the mortality rate was approximately eight per cent, they added. Respiratory and heart and blood vessel complications were most commonly noted in the fatal cases.

There were no complications following 190 operations in the larger group, the doctors stated. When complications occurred, the commonest were those affecting the respiratory system, the surgical wound, and the heart and blood vessels. "Considering the age of the patients in these studies and the severity of their diseases, one must conclude that the surgical operations were well tolerated."

In considering the problems of preoperative and postoperative care of the elderly, "one is impressed with the point that they differ only in degree as compared with the surgical care of younger patients," the doctors stated. The margin of safety is less, and sharper attention to details is necessary. Expert nursing care is a potent factor in recovery. They added that the daily routine of the elderly surgical patient should be upset as little as possible, and that he should be returned to his normal environment and activities as soon as practicable.

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THE ROLE OF THE GENERAL PRACTITIONER IN THE PREVENTION OF BLINDNESS

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Next to life itself the greatest single asset the individual possesses is sight. Only one organ in the body, the heart, functions more constantly than the eye. There is no single examination which can give you as much information about the body as an examination of the eyes. This is the only place in the body where you can see the progress of a pathological lesion without a biopsy.

The general practitioner should make a routine visual record of every new patient who comes in for examination. Along with the routine visual examination should be a routine Wassermann test. One of the late complications of neurosyphilis, optic atrophy, could be prevented if treatment were given in an early stage of luetic infection.

GLAUCOMA

Of the 314,000* people blind in the United States, one-eighth of them are due to glaucoma. The general practitioner should inquire into the history of glaucoma before prescribing certain drugs, such as atropine or instilling any mydriatic for fundus study. Glaucoma has resulted from giving atropine and its alkaloids by mouth for various general conditions; digitalis and quinine have also been known to effect the vision.

DRUGS FOR REDUCING DIETS

Various drugs which are advertised in the lay press for reducing diets have in many instances effected the nutrition of the eye and in some cases resulted in amblyopia. One drug which has been used to reduce weight is dinitrophenol which, in some instances, has resulted in the formation of cataracts. Before any drug is prescribed to reduce weight it would be wise to consult an ophthalmologist as to possible detrimental effects on the eyes.

DIABETES

Eye complications resulting from diabetes are cataracts, iritis, retinitis and hemorrhage. These complications, in many instances, can be prevented and controlled by an alert general practitioner. Diet and insulin are the only means of preventing these complications. Every diabetic patient should be under the continued observation of a competent ophthalmologist. In these cases the knowledge of the general practitioner and the ophthalmologist should be complementary.

MALIGNANT EXOPHTHALMOS

Malignant exophthalmos has sometimes resulted from thyroidectomy in women of the menopause age who show eye signs but few systemic signs of hyperthyroidism. Any practitioner who is called to give anesthesia should try to avoid undue pressure on the eye; it can result in central retinal occlusion and corneal abrasions. Care should be used to avoid accidental dropping of ether in the eye; this can cause severe damage to the corneal epithelium.

OBSTETRICS

Congenital anomalies of the eye may develop sometimes if the mother sustains even a trivial illness during the first trimester of pregnancy. If a patient develops German measles during this critical period, abortion should be considered. Retinal detachment is one of the complications of pregnancy and every pregnant mother should have her eyes examined periodically by a competent ophthalmologist. The care of the eyes of the newborn by both the doctor and the nurse is most important. They should never forget the prophylactic measures in the prevention of ophthalmic neonatorum by the instillation of silver nitrate. Two and eight-tenths per cent of blindness in children is caused by prenatal syphilis. The Wassermann test should be a part of the routine examination of every pregnant woman. If positive, prompt and energetic treatment should be instituted. All premature infants should have their eyes carefully examined; doctors and nurses should be on the alert for a condition known as retrolental fibroplasia which occurs in 15% of these infants.

PEDIATRICIAN

The pediatrician should observe the motility of the infant's eyes at an early date and if there is any indication of crossed eyes or a squint they should be referred to an ophthalmologist. The squinting eye should be treated before the child is three years of

*Read before the annual meeting of The Medical Society of Virginia at Roanoke, October 18-21, 1953.

age. After that time definite organic changes take place in the eye and vision is usually lost in the squinting eye. In acute infectious diseases, such as measles and chicken pox, the child's eyes should be carefully guarded and, if there is any suspicion of corneal ulcer or eye complication, the ophthalmologist should be consulted.

How People Become Blind[2]

Children

Defects of Prenatal Origin, Cause Unknown ---- 49%
More and more babies are being kept alive, but some of these infants escape death only to become blind from diseases, such as retrolental fibroplasia.

Infectious Diseases ---- 14%
Most serious is syphilis. An estimated 100,000 babies face blindness because of syphilis.

Heredity ---- 16%
Main problem is to educate people with hereditary blindness concerning the dangers of passing the defect on to their children.

Injuries ---- 7%
An estimated 90,000 eye accidents occur annually among school children. About 1,000 of these accidents cause a child to lose the sight of one or both eyes.

Others ---- 14%
Includes tumors, general diseases, poisonings, and unknown causes.

Adults

Cataracts ---- 17%
About 49,000 Americans are blind from cataracts, which is a cloudiness of the lens of the eye. Many of these people could see again with the help of surgery, but are unaware that they can be helped.

Glaucoma ---- 12%
This disease, which usually strikes after 40, causes the eyeball to harden. The resulting pressure destroys the optic nerve and sight is lost. Early treatment is vital to save sight.

Infectious Diseases ---- 23%
Syphilis is most frequent. But the new "miracle drugs" now make it possible to cure cases in a short time. Provided treatment is adequate and started early, blindness may be averted.

Injuries ---- 9%
About 300,000 eye injuries occur each year in industrial plants. Thousands of others happen in homes, and on farms.

General Diseases ---- 6%
This category includes diabetes and high blood pressure.

Others ---- 11%
Including poisonings, tumors, defects of prenatal origin.

Unknown ---- 22%

How Do We Stand in the Fight Against Blindness?

Blindness is being defeated on some fronts, but is gaining on others. What are our chances for good sight?

Among children there has been:

A 54% drop in blindness due to venereal disease since 1936.

A 96% reduction in blindness from "babies' sore eyes" since 1908.

A 30% decrease during past 14 years in blindness due to injuries.

But, on the other hand, since 1936 there has been a sharp increase in blindness from prenatal and hereditary causes. This is mainly due to retrolental fibroplasia, a disease of unknown origin which usually strikes only premature babies. It now accounts for more than 50% of blindness among preschool children in some states.

Among adults, the number of sightless is increasing at the rate of 4,800 each year. The reason: more people are living longer as medicine stretches the life span, and more are stricken by blindness diseases that usually occur in later life.

Blindness Is Increasing, Yet More Than Half Can Be Prevented

Every 24 minutes someone goes blind! Today there are more than 260,000 blind Americans. Another 1,000,000 men and women are blind in one eye; and thousands of Americans have vision that is only barely useful.

Sixty Americans become blind each day; 420 each week; 22,000 during each year. Yet more than half of these men and women should be able to read this page as clearly as you! Medical science now has the knowledge to prevent at least one out of every two cases of blindness. But this knowledge is worthless if it is not put to use.

How We Can Prevent Blindness

I. *Regular Eye Examinations for School Children*

Children seldom complain about poor vision because they don't know how well they ought to see. It's up to parents and teachers to know the symptoms of eye trouble and to arrange for a professional examination when indicated. Watch for these signs of visual defects in children:

Rubs eyes often, blinks more than usual, frowns, shuts or covers one eye. Has difficulty reading or

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doing other close work, inflamed eyes, red-rimmed or swollen lids, recurring sties.

II. *Guard Against Accidents*

Most blindness due to accident can be prevented! Example: Children most frequently suffer eye injuries while playing unsupervised games. Here's what you can do: keep dangerous instruments, such as scissors, knives, etc., away from children. See that youngsters do not take chances with dangerous "toys" like BB guns, slingshots, bows and arrows. If you work in an industrial plant or have a home work-shop, remember that one bit of flying wood or metal can cause blindness. Wear safety goggles wherever there is a chance of eye accident!

III. *Take Proper Care of the Eyes*

Healthy eyes never "wear out." You can read or watch television as much as you like without ruining your vision. But your eyes become tired quickly if not used properly. Shade light at eye level so that it is diffused without glare. Make sure your television set is adjusted correctly, and don't sit too close to the set.

Never rub your eye, especially if there is a foreign body in it. Pull down the upper lid over the lower and let the tears wash away the speck. If this doesn't do the job, wash the eye with luke-warm tap water. See your physician if the speck remains.

One Out of Four Children Needs Eye Care, yet many of these 6,500,000 children are not being treated because of neglect.

Sixty thousand American school youngsters need special help to keep abreast of their normally seeing classmates. This help includes facilities such as books printed in large size type, special desks and other equipment. Still there are over 52,000 children who need these aids and don't have them.

An estimated 90,000 Eye Accidents Occur Annually Among American Youngsters. About 1,000 of these cause the child to lose the sight of one or both eyes. Boys have three times as many eye mishaps as girls; junior high school age is the most dangerous.

Safety engineers estimate that 90% of the 300,000 annual eye accidents in industry can be prevented. Each year thousands of men and women are saved from blindness simply because safety goggles and other eye protection were worn when necessary.

Poor Vision Hampers U. S. Defense Program. Poor vision is a "fifth column" sabotaging America's defense effort. Defective eyesight costs our country about 7% of her military manpower, and slows down the production efforts of nearly 40% of her industrial workers. During the last war, the enlisted men for about 20 infantry divisions were lost to the U. S. Armed Forces because of poor sight. On the production front, four out of every ten workers in small plants have a vision defect that reduces their efficiency. Poor vision is an enemy; guard against it.

IV. *Periodic Eye Checkups—Especially After 40*

After 40 cataract and glaucoma, two major eye diseases are leading causes of blindness. Early treatment is important, especially in glaucoma. That's why regular, periodic checkups are so important. Body ailments, such as diabetes, which affects your eyes, also occur most often in later life. After you reach 40, an ounce of prevention is worth more than a pound of cure—it is often the difference between good sight and blindness.

What Blindness Costs the U. S. An estimated $150,000,000[4] is expended annually in educating and caring for the blind. In addition there is the cost of compensating industrial workers for blinding eye accidents. An estimated 80,000 men and women have lost one eye in industrial accidents, and another 8,000 are completely blind from injuries. The total cost to industry—in compensation, medical expenses and lost production—is well over $200,000,000 a year. Of course, the full cost of blindness in terms of human suffering can never be calculated.

800,000 Slowly Going Blind, But Don't Know It. Over 800,000 men and women over 40 in this country are slowly being blinded by glaucoma and don't know it. Symptoms may be so mild that the patient feels no pain at all. If glaucoma is found early, blindness can usually be prevented. There is no way to restore sight lost through glaucoma, but the progress of the disease can be halted. When treatment is delayed, sight may be lost which can never be regained.

Preventing Blindness Is Everybody's Job. Saving sight is a job for everyone—the parent, worker, and businessman—as well as the doctor, nurse, social

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worker, teacher and public health officer. There is need for better vision testing of school children, for more effective ways to reduce accidents, and to find and help those in need of eye care. And the need is for an intensified program of public education, and more research into the causes of blinding eye diseases.

What U. S. Spends to Prevent Blindness. Despite the huge cost of blindness, less than $500,000 a year is spent by the United States for organized prevention of blindness activity. And less than one million dollars a year is spent for research on eye diseases. All told, America spends a total of about one-and-a-half million dollars annually to fight blindness. Compare this to the following statistics:

Over-the-counter eye lotions	$ 2,625,000
Eye cosmetics	5,095,000
Over-the-counter headache relievers	101,510,000
Candy and chewing gum	1,854,548,000
Tobacco products and supplies	4,124,467,000
Alcoholic beverages	8,800,000,000

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Unusual Group of Symptoms in Three Patients Described.

An unusual group of symptoms, consisting of recurrent episodes of unnatural drowsiness, excessive sleep, extreme hunger and such abnormal mental states as depression, clouded senses, partial or total amnesia for certain periods, and psychomotor retardation, were described in the March 27 Journal of the American Medical Association.

The occurrence of such symptoms in three patients was reported by Dr. Alfred Gallinek, New York. In two of the patients, the symptoms followed an acute infectious illness.

One case described by Dr. Gallinek was that of a 58-year-old woman who began experiencing the symptoms following an acute attack of diarrhea in Haiti 12 years ago. The episodes occur two or three times a year, lasting for about three weeks.

First the patient becomes excessively hungry, with a special craving for sweets. She then experiences extreme drowsiness, bodily discomfort, fatigue, weakness, and sleeps 18 to 24 hours each day. When awake during this period of the attack, the patient shows slowness of speech and thought and a reduction of all motor activity. The third phase of the attack is characterized by abnormal mental states.

The second case reported was that of a 34-year-old man who has experienced similar symptoms since he was 17 years of age. The third patient was a 16-year-old youth who began suffering from such symptoms following an attack of chickenpox and subsequent brain fever at the age of 14 years.

Drug therapy during the last year has resulted in a very remarkable improvement in the older patients, according to Dr. Gallinek. Although the episodes have continued, they occur at longer intervals and are much milder. No attempt at therapy has been made in the case of the young boy.

Dr. Gallinek is associated with the department of neurology, Columbia University College of Physicians and Surgeons, and the neurological institute of the Presbyterian Hospital of New York.

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NOTES ON PULMONARY TUBERCULOSIS*

Standard Diagnostic Study (III) Tuberculin Test (B)

THE VOLLMER PATCH TEST: *Method of Application:*

A square of filter paper saturated with Old Tuberculin (O.T.) is applied to the skin supported by gauze and a strip of adhesive tape. Usually a surface is selected in the region of the upper sternum or between the scapulae. The skin surface should be cleansed and defatted with acetone. The filter paper should remain in contact for 48 hours, and the test read after *an additional* 48 hours. The subject (or parent) must be specifically instructed to remove the patch 48 hours after it is applied—warned *not* to permit it to remain until the test is normally due to be read. (96 hours of continuous contact has been known to cause unnecessarily severe inflammatory reactions). The subject (or parent) should be instructed that should the area become irritated to a point of real annoyance before the expiration of the first 48 hours he should seek his doctor's advice with respect to earlier removal, or if a physician can not be contacted, to remove it himself, when in doubt.

Interpretation: A positive reaction shows redness, infiltration, closely set elevations and depressions upon the skin area to which the square containing tuberculin had been applied. There will seldom be vesiculation *provided* the patch is removed 48 hours following application.

Advantages: More easily applied (without use of needle); singularly well adapted therefore for children and for the testing of single individuals in office practice.

Properly applied and with good cooperation the test can be only slightly if at all less accurate than the Mantoux (intradernal test of 1:10,000 O.T.). Patches are stocked by the Bureau of Biologics, Virginia State Health Department, and are supplied upon request and without cost to private physicians and health departments. They should be kept in a cool place until used but need not be refrigerated; expiration date on the patch test should be noted—this *can* consist of a maximum of 18 months.

Disadvantages: The amount of tuberculin coming in contact with the skin is not dependably uniform. The test is subject to the cooperation of the person being examined. If the patch is removed too early, becomes loosened or wet, the reading will be inacurrate.

There is only the one strength test (See April issue, *Virginia Medical Monthly*); if negative, higher concentrations of tuberculin must then be introduced intracutaneously, when the tuberculin test is employed for differential diagnostic study.

THE MANTOUX TEST: *Method of Administration.*

The Mantoux test is administered intracutaneously using either O.T. or P.P.D. A "tuberculin syringe" should be used; this has a blue plunger and is graduated in tenths of a cubic centimeter. A short bevelled 26-gauge, 3/8 to 1/2 inch steel needle is standard, for individual patients; a separate needle can well be used for each patient if the group is not too large. The syringe (and needle) should be initially dry (to prevent dilution of tuberculin particularly when only .1cc is drawn into the syringe; the drawing of multiple doses of tuberculin into one syringe for multiple injections without refilling is generally advised against.)

Needles and syringes, and ideally the sterilizing equipment, used for tuberculin testing, should not be used for or in connection with other types of injections. Minute amounts of tuberculin which remain in the needles and syringes even after repeated sterilization may render other types of tests invalid.

The flexor surface of the mid-upper forearm is cleansed with acetone or alcohol. When the skin is dry, it should be rendered firmly taut; then inject exactly 0.1 c.c. of whichever type and strength tuberculin is being employed, into the upper layer. Care must be taken not to penetrate under the skin. When the injection is really *intra*-cutaneous, it produces a small raised bleb or wheal, which moves with the

*Prepared by the Virginia State Health Department.

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skin; otherwise the material has been injected *under* the skin and the test will be unsatisfactory. Inject exactly one-tenth cubic centimeter; do not guess at the amount from the size of the wheal produced by the injection. Needless to say, do not give a second test immediately following a full dose that has been administered too deeply; the patient's reaction to the latter should be observed before a second attempt is made (48 or more hours later) to inject the tuberculin *intra*-cutaneously.

Interpretation: Customarily, a reading is recommended to be made after 48 *or* 72 hours. One naturally questions whether there *can* be *two* such widely divergent *optimal* times to read a tuberculin test. There *can* be, and *is,* a difference of opinion as to what is the optimal period.

The Virginia State Health Department generally, uses the 48 hour period. *After* 48 hours positive tests tend to fade, especially those of 3 plus and under; accordingly a 1 plus test *may* be completely missed when a later reading is made.

If for any reason the individual cannot be seen at the end of 48 hours, he should, if possible, be contacted within 96 hours of the time of injection, for the reaction, though declining, *may* be visible for several days past the optimal time for reading. A negative test under these circumstances would call for a repeat test, especially if the individual himself had noted some reaction at 48 hours.

The forearm should be in good light and flexed a little at the elbow . (If held fully extended, the muscles may be taut enough to obliterate evidence of a positive test.)

The positive reactor has an induration edema of at least 5 mm measured in its widest diameter. *The presence of redness or erythema is not significant unless associated with edema.* When the induration (edema) is absent, the reading is negative; when there is a trace of induration, less than 5 mm in diameter, the reading is negative or at the most "doubtful".

In evaluating a reaction it is well to look across the arm rather than down on it. Pass a finger over the tested area. Induration caused by the edema can sometimes be felt when it does not produce an elevation that can be seen.

Positive reactions are recorded (arbitrarily) as follows:

one plus = 5-10 mm. induration
two plus = 10-20 mm. induration
three plus = Exceeding 20 mm. induration
four plus = Extensive edema with central necrosis

(Very rarely, instead of or in addition to edema, vesiculation may occur)

When test is negative or "doubtful", retesting with the same or higher concentration can be done immediately, or after any convenient interval.

Comparison Table of Strengths:

O.T.	P.P.D.
1:100 (100 times stronger than routine initial dose) =	0.005—2nd strength *250* times stronger than 1st strength
1:1,000 (10 times stronger than routine initial dose) =	0.0002—intermediate strength (10 times stronger than 1st strength)
1:10,000 (routine initial dose) =	0.00002—1st strength

(The Patch Test is roughly equivalent to 1:10,000 O.T.)

Initial Dosage: Ideally, the fewer tests required to establish the suspect's tuberculin status the better. But one is obligated to take all reasonable precaution against unduly severe response, not only because of the inconvenience of a 4 plus reaction locally (skin), but to protect against a potentially harmful *focal* reaction which also *may* occur in the lung (or other organs) where the patient is highly allergic.

It stands to reason that the higher the initial dose the more often one will observe a 4 plus reaction.

However, from a practical standpoint, neither the 1:10,000 nor the 1:1,000 O.T. strengths produce 4 plus reactions often enough to serve as a deterrent to their use as the initial test strength, where no known contraindications exist. Where an x-ray has preceded the tuberculin testing, as happens almost invariably in adults (suspects and close contacts) the initial dose can be reduced to 1:100,000 if the pulmonary lesion appears to be a little more than moderately acute.

In contrast, in children, (suspects and close contacts) due in part to technical difficulties in obtaining a satisfactory film, x-rays are usually restricted to those having a positive tuberculin reaction. Accordingly, except for the comparatively rare instances where the suspect child's lung can conveniently be screened fluoroscopically prior to tuberculin, test, the initial test had best not be stronger than 1:10,000,

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or preferably might consist of a patch test where good cooperation can be anticipated; as noted above the patch can be removed early if an excessive reaction occurs.

Particularly should the patch test (when removed early) or a 1:100,000 O.T. Mantoux be employed in children having swollen lymph glands, ulcerations or discharging simuses or other extra-pulmonary forms of disease.

Contra-indications (General): Tuberculin testing of any strength or type should not be attempted:

1. In acute illness
2. In diseases of the skin.
3. Concurrently with smallpox vaccination.

"False Negative" Reactions (where tests have been properly administered) are sometimes seen:

1. In acute (toxic) pulmonary tuberculosis
2. Early in tuberculcus effusion.
3. During concurrent exanthemata.
4. During the early anergic phase of primary infection. Because 6-8 weeks are ordinarily required for a tuberculin test to become positive following infection, the State Health Department recommends that a child, who has been in close contact with an active case of pulmonary tuberculosis, should be tuberculin tested a second time 2-3 months following break in contact, if the first tuberculin test taken during the potentially anergic stage has been negative.
5. Known positive tuberculin tests have been observed to revert to negative in a small percentage of persons who have never had, or who no longer have clinically active tuberculosis.
6. A negative tuberculin test, including higher concentrations, is reported with extreme rarity upon persons with clinical tuberculosis even with a positive sputum, and where the disease is not acute. This seeming paradox should not be permitted to undermine one's confidence in the tuberculin test for use in the many *many* instances where its application is of tremendous, not to say crucial importance; rather had this exception better be cited as merely one more example of an age-old adage to the effect that the words "always" and "never" have no place in a physician's vocabulary, with respect to *any* disease!

New Bcoks.

Below are given names of some of the newer books in the Tompkins-McCaw Library of the Medical College of Virginia. These may be borrowed under usual library rules.

Biggs and Mcfarlane—Human blood coagulation and its disorders. 1953.
Burrow—Science and man's behavior. 1953.
Cole and Elman—Textbook of general surgery. 6th edition, 1952.
Crohn—Regional iloitis, 1949.
Current therapy. 1954.
Fisher and Hawley—A few buttons missing. 1951.
Hauser—Diseases of the foot. 2nd edition, 1950.
Keys, et al—Biology of human starvation. Vols. I and II. 1950.
Memoirs of the society for endocrinology—The determination of adrenocortical steroids and their metabolites. 1953.
Memoirs of the society for endocrinology—The thyroid gland. 1953.
Methods in medical research. Vol. 6, 1954.
Moloney—Understanding the Japanese mind. 1954.
Murphy—Heredity in uterine cancer. 1952.
Podolsky—The jealous child. 1954.
Podolsky—Music therapy. 1954.
Potter—Fetal and neonatal death. 2nd edition, 1949.
Russell—Poliomyelitis. 1952.
Slater—Psychotic and neurotic illnesses in twins. 1953.
Yorke—Salt and the heart. 1st edition, 1953.

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MENTAL HEALTH

Joseph E. Barrett, M.D.
Commissioner, Department Mental Hygiene and Hospitals

Hospitalization as a Corrective Experience*
(The Story of Mrs. Jones)

Virginia has the distinction of being the first state to have a hospital totally for the care of the mentally ill. The philosophy at that time was for confinement. Today the aim is for the rehabilitation of over 80% of the patients. This is calculated to be the number that, with present knowledge, can be helped to lead useful lives again.

Great advances have been brought about in physical medicine because of the emergencies created by the recent war conditions. Rapid advances were also made during the last war in the treatment of mental patients. Some of these include narcoanalysis, group therapy, together with refinements of other techniques in caring for patients. With the shortage of labor created by the war, Virginia hospitals, like all other mental hospitals in the United States, were faced with a dire shortage of personnel. Patients had to be used in increasing number to perform necessary functions to keep the hospitals running. It was noted that many of the patients improved greatly when they were given some useful tasks to perform. Out of this grew the concept of hospitalization as a corrective experience.

Many of the patients at Southwestern State Hospital have become ill because they were unable to face the demands of living at home. As life became too difficult for them to face, they withdrew from the reality of everyday living and either developed strange ideas or physical complaints when there was no organic basis for these complaints. It was the hospital's job to guide them back on the road to recovery.

To demonstrate how a hospital can set up an environment which serves to correct the previous harmful experience of the patient, I would like to tell you a story of Mrs. Jones. Mrs. Jones is not the real name of a patient at Southwestern State Hospital, but there is a patient at Southwestern State Hospital whose story this belongs to. Mrs. Jones was born in a small coal mining town in West Virginia. It was necessary that she and her mother live with relatives who had children of their own. As a small child, Mrs. J. felt that she didn't belong here and that this really wasn't her home. Her uncle was a man who had strait-laced ideas on how children should be brought up. To him life was very real and earnest. Even children should not be allowed to have the frivolities of having a good time. When Mrs. Jones entered adolescence, she and her mother moved to Virginia to live with another relative. Here, too, our patient was closely supervised. She was not allowed to be out in the evening or to have dates with boys. However, Mrs. Jones managed to escape and to surreptitiously occasionally have dates. She married an immature man who had difficulty holding a job. Mrs. Jones and her husband had four children and the children were a source of much pleasure to Mrs. Jones. She worked to support herself and the children and received some assistance from her husband's family. Many of the jobs that she had were in factories which had the "piece-work" system. Mrs. Jones had to be good, she had to complete a certain number of projects, and each task had to be meticulously performed. She would check it and re-check it to make sure it was right. She could not tolerate the slightest imperfection. Of course, she was unable to keep up the production schedule and was discharged. She, however, did get a job in a laundry and was able to accomplish at this task and to do satisfactory work in which she received gratification as well as money to support her family. Her husband died a horrible death in another state. His body was so mutilated that it was impossible to recognize him except for identification papers which he had on his person and also several tattoos.

Mrs. Jones was haunted with the question "was this really her husband they had buried?" It was impossible for her to realize that he would not again return to her home and knock on the back door, to be allowed to enter as he had done so many times before. "Was she in some indirect way responsible for his death?" "Could she have changed him?" "Did she take care of him as she should?"

It became necessary for Mrs. Jones to stop working and take care of her children. One of her

*Article prepared by Thomas H. Lahey, M.A., Staff Psychologist, Southwestern State Hospital, Marion, Virginia.

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children became ill and had to be hospitalized. She also incurred heavy expense in necessary improvements to her home. Faced with the inability to earn her living and family worries, Mrs. Jones became increasingly depressed. She could not sleep at night and lost weight. Mrs. J. became obsessed with the thought that she was going to kill her youngest child. She was unable to hold the child and take care of her because when she was near her, the thought would enter her mind. She was panicked by the thought that she would carry out her ideas. The situation became so difficult and Mrs. J. was so upset that she was hospitalized at Southwestern State Hospital. On admission, Mrs. J. received a thorough physical examination and was seen by the psychologist and the psychiatrist. A plan was set up to help her on the road to recovery. Because she had succeeded so well at her job in a laundry, she was given a similar job in the Hospital Laundry. The employee supervisor of the laundry praised Mrs. J. for her work and Mrs. J. received much gratification from performing a task. No pressure was put on her to accomplish. She was seen twice a week for interviews by a member of the staff and it was pointed out to her that she was performing a useful task for the hospital and that she should not feel obligated to the hospital for the care which she was receiving. Her family difficulties were discussed with her and gradually she came to understand herself and to function more adequately. Initially, she wore her hair in a very severe bob and wore dark clothes and no make-up. She was encouraged to wear whatever type of clothing she liked and she soon asked the hospital beautician to give her a permanent. She learned how to use make-up correctly and started attending the recreational activities. She enjoyed the movies and the dances and she was consequently given approval to attend these functions. A relative gave her a small amount of money and she was allowed to go on a shopping spree. She bought the material for a dress. She selected a red dress and some dress shoes. She has increasingly become more relaxed and has found out that, although there are difficulties in almost everyone's life, life in general can hold a certain amount of pleasure also and it was not wrong to have a certain amount of fun. Mrs. J. has learned to smile and now takes delight in coming and telling of comical incidents that happened to her at the hospital. She is regaining confidence that she can leave the hospital and support her children and earn her own living. She no longer is possessed with the fears that she is going to injure her child and when the child was brought to see her she was able to play with her and enjoyed her very much.

This is only one example of how the State Hospital serves as a corrective experience for the total individual. A visit to the hospital will show many patients who are able to work at certain jobs and receive gratification from them. Some of them will be working on the hospital farm, others will be helping in the food service. In the diagnostic and admission building, you may be met by an attractive and courteous middle-aged woman who functions as a receptionist. You would not realize that this woman is a patient at the hospital and only a few months before was very withdrawn and was a severely emotionally upset and psychotic patient. She is now on the road to recovery. The receptionist in the physical medical building is also a patient. To be sure, these patients are supervised by an employee who keeps a friendly watch over them and helps them to see that they can function adequately and can be a useful member of society. All of the employees have an obligation to help the patients become more adjusted and useful citizens, besides taking their part in the task of operating the hospital and carrying out their routine duties. Some of the patients are members of a garden club class which is sponsored by the Marion Garden Club. They plant flowers, watch them grow and make flower arrangements. Some of them, at first, do not actively engage in the projects but only watch and as they are constantly encouraged to try their hand at a variety of tasks; gardening, being a receptionist, clerking in the canteen, many of them finally are able eventually to leave the hospital and take up their lives in the community.

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PUBLIC HEALTH

MACK I. SHANHOLTZ, M.D
State Health Commissioner of Virginia

Typhoid Fever Carriers

Revision of the list of typhoid carriers in Virginia has just been completed and 118 known carriers, involving 51 counties and 5 cities, have been registered from the various health departments throughout the state. The number of carriers has not changed materially in recent years, as evidenced by the fact that the inquiry sent out in 1950 brought in reports on 112 and those in 1952 and 1954 revealed 122 and 118 respectively. During the interval years additions to or subtractions from the list are made according to reports received from local health departments. Results of the annual examination of the stools of known carriers and of those who had the disease as long as one year previously are important in making a roster of typhoid carriers.

The control of typhoid fever in the state is largely dependent on the detection and supervision of this group which serves as the reservoir for typhoid organisms. The transmission of typhoid fever is through the fecaloral route and this chain must be broken if these people are to be prevented from spreading the disease.

Chloramphenicol has not proved helpful in destroying the bacteria in carriers. It is stated that cholecystectomy will eradicate the carrier state in about 90% of cases; however, the most successful means of breaking the link has been through supervision and education. Carriers should be taught that they have a chronic infection which they may transmit or carry to other people and that they have a responsibility in preventing this. They must be taught the rules of personal hygiene. They must be warned that they are not to handle food, milk or drink except in their own homes, and only for themselves or their immediate families who are known to have been immunized against typhoid fever. They must be advised to report any change of residence so that they may be listed in the files of the local health authority whose jurisdiction they are entering. Families of carriers must cooperate in all of these measures and assume responsibility for maintaining their own immunizations.

During the past five yeasr, the cases of typhoid fever reported in the state have been as follows:

1949	1950	1951	1952	1953
116	68	61	77	54

The reduction in the incidence of typhoid has been brought about through community efforts in establishing and maintaining environmental sanitation that insures safe water supplies, providing sanitary sewage disposal, and regulating the production and distribution of milk. Pasteurization of all milk is important in the control not only of typhoid fever but of many other diseases.

While there are fewer cases each year, this does not mean that we can relax in our efforts to prevent its spread. Except for those who have had the disease and for the group which maintains adequate protection through the use of vaccine, the susceptibility of the general public remains unchanged. We must remember that the disease is not eradicated and that any break in the barrier of protection that we have set up will result in its reappearance in the areas involved.

MONTHLY REPORT OF THE BUREAU OF COMMUNICABLE DISEASE CONTROL

	March 1954	March 1953	Jan.-March 1954	Jan.-March 1953
Brucellosis	3	6	7	15
Diphtheria	2	15	19	44
Hepatitis	699	143	1695	570
Measles	4426	739	7306	1482
Meningococcal Infections	15	27	38	87
Poliomyelitis	6	2	12	7
Rocky Mt. Spotted Fever	0	0	0	0
Streptococcal Infections (Including Scarlet Fever)	508	540	1619	2144
Tularemia	0	3	16	14
Typhoid Fever	4	3	10	9
Animal Rabies	48	56	126	152

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MEDICO-LEGAL NOTES

Air Embolism*

Death from air embolism is a tragic catastrophe which has been reported following numerous injection procedures (tubal patency test, pneumothorax, pneumoperitoneum, ventriculography, intravenous infusions, perirenal insufflation, etc.) as well as during thoracic and thyroid surgery. Perhaps it is seen most frequently by the forensic pathologist after attempted criminal abortion when large amounts of air enter the venous circulation as frothy bubbles injected into the uterus as a soap solution. It has even been described following Caesarean[3] and normal vaginal delivery.[6,7] We have seen one case in our office after a spontaneous abortion.

The forensic pathologist must keep this possibility constantly in mind, lest the opportunity to demonstrate the phenomenon be forever lost by hindsight rather than proved by foresight. Probably everyone is more or less familiar with the more common form of venous or pulmonary air embolism. Here, relatively large amounts enter the venous circulation and are carried to the right heart and thence the pulmonary artery circulation. Relatively large amounts of air (100-150cc) are needed to produce death and the rapidity of entry is of the utmost importance.[4,5,8] Other factors determining the fatality are position and tachypnea. With the body positioned on the left side, air within the right ventricle is trapped away from the pulmonary artery allowing the pulmonary circulation to continue until the air is gradually absorbed. Knowledge of this mechanism can be life saving in some cases. The increase in respiratory rate may serve as an excretory mechanism for the entrapped air, since experimental work has demonstrated that in open chest observation with artificial breathing (preventing tachypnea), small amounts of injected air were consistently fatal.[4] It is claimed that if large amounts of air enter the venous circulation slowly, the air may pass the lung barrier and embolize to the coronary artery.[8] However, in the vast majority of cases, entry is sufficiently rapid to cause death from pulmonary block. Such factors as vasovagal reflex and reflex coronary spasm has been ruled out experimentally by previous vagotomy, which failed to influence the results as compared with animals with intact vagi.[1]

In sharp contrast to venous embolism is the arterial type where air gains entrance to the left side of the heart via tears in the lung or pulmonary veins and then may embolize to the coronary or cerebral vessels. Besides extrinsic sources (injected air, air aspirated into pulmonary veins during surgery), intrinsic air in the lung itself is a potential source of danger. The establishment of a broncho-venous tear by such simple trauma as needle puncture of the lung or tearing of the lung substance by an adhesion during collapse therapy may serve as the portal of entry of air from the lung itself into the pulmonary vein. Most cases of so-called "pleural shock", occurring during attempted fluid aspiration when no air injection is involved, are probably in reality due to the above mechanism. The rarity of such accidents when dealing with normal lung suggests that pulmonary infiltration may be a needed factor, the diseased tissue preventing closure of these accidental communications.[4] Another mechanism of intrinsic arterial air embolism may occur in free escapes from diving or submersive apparatus. Here the victims fills his lungs with air at a greatly increased pressure beneath the surface and rises without breathing device to the top of the water. If he fails to "blow out" as he rises, the increase in air volume within the alveoli as the water pressure decreases may rupture the alveoli and small pulmonary veins allowing air under high pressure in the lung to enter the left side of the heart. These people sometimes die within minutes after being removed from the water, possibly from ventricular fibrillation secondary to myocardial ischemia from blocking of the coronary arteries with air. Again in sharp contrast to the venous type are the amount of air needed to cause death and the clinical symptoms. Kent and Blades (cited by Durant *et al*[4]) found that 1 cc. of air injected into the pulmonary vein of twenty-eight dogs was uniformly fatal. Other workers found severe ischemic disturbances of the myocardium by E. K. G. when .025 cc. of air was injected directly into the left anterior coronary artery of dogs, and that .05cc was often fatal.[4] Ventricular fibrillation was often observed. Therefore we may assume that in this form of air embolism, far smaller

*Contributed by: George W. Thoma, Jr., M.D., Assistant Chief Medical Examiner, Office of the Chief Medical Examiner, Richmond, Virginia. (References available on request).

amounts are apt to produce serious effects. The clinical picture will depend on the localization of the emboli, whether cerebral or coronary, and this to a great extent will be determined again by patient position. In addition to the localizing symptoms, such findings as air bleeding from cuts in the skin, marbling of the skin, and air in the retinal vessels are described. Chase[2] reported a case of cerebral air embolism in which stereoscopic x-rays of the skull taken within three minutes of death following cutting of a pulmonary vein during lobectomy revealed air in the Circle of Willis and the middle cerebral arteries. He also demonstrated experimentally that air acts as an irritant to produce vasospasm, further aggravating the mechanical block and possibly persisting after the air is dissolved.

The technique of post-mortem demonstration of venous air embolism is relatively simple and well known to all practicing pathologists. However, the demonstration of air in the left side of the heart or the coronary or cerebral vessels may be more difficult because much smaller amounts may be present or the possibility may not be thought of. The following technique as done in our office is suggested as a routine for all sudden unexplained deaths as well as in all suspected cases of air embolism. Of course, post mortem gaseous decomposition or gas bacillus infections invalidate all this and must be ruled out by the remainder of the autopsy and culture if necessary. The sternum and costal cartilages are exposed by a simple midline incision starting from below the sternal notch so as to avoid the superficial neck veins and the skin and muscle are carefully reflected laterally. The rib cartilages are cut with rib shears up to but not including the first rib. The freed sternum can then be lifted upward and broken across the angle, exposing the pericardium. The aorta is tightly ligated proximal to the origin of the great vessels and the pericardium is opened and flooded with water to submerge the heart. A single superficial cut across the coronary arteries, care being taken not to enter the chambers, followed by gentle finger pressure along the course of these vessels will demonstrate air bubbles rising to the surface if present. Then the pulmonary artery, right ventricle, and right atrium can be incised separately and gentle pressure exerted to release any trapped pockets of air . In like manner the left ventricle and auricle are opened. Special care must be taken in examining the brain. Air bubbles can often be observed as they are sucked into the cerebral vessels from the severed neck vessels with traction on the brain during removal. Therefore it is essential that no neck vessels be disturbed beforehand and that the aorta be securely ligated. After removal of the calvarium (caution is necessary here to avoid cutting the underlying brain with the saw), gentle traction on the frontal lobes exposes the internal carotid arteries which are clamped or ligated and then cut proximally. The basilar artery is similarly treated, and the brain removed. If air bubbles are not visible in the Circle of Willis or the immediate branches, the brain is submerged and each clamp removed separately and the vessel end observed for their appearance. It is only by rigid adherence to such a technique and a high index of suspicion in all unexplained deaths that the frequency of such diagnosis as "unknown, pleural shock, vagal reflex, etc.," can be reduced and the true cause sometimes found.

Coffee a Stimulant, with or without Cream.

You can drink your coffee with or without cream without affecting its stimulating qualities—if you can afford coffee at all at today's prices.

In reply to a query to the Journal of the American Medical Association as to whether black coffee has any more stimulating effect on the nervous system than coffee with cream, a medical consultant wrote:

"No. Black coffee may be a little more concentrated, but the addition of cream does not alter its chemical composition."

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THE MEDICAL SOCIETY OF VIRGINIA

Report of the Committee on Legislation

Although the recent session of the General Assembly was a stormy one in some respects, the legislation proposed by the Society and supported by its Legislative Committee had rather smooth sailing and came through without too much difficulty. This result was due in part to the subject matter of the several bills presented, and in part by the splendid assistance given your Chairman by Dr. W. C. Caudill in the Senate and Dr. W. C. Elliott in the House.

LICENSING OF FOREIGN GRADUATES.

At the annual meeting of the Society in October a special committee, with Dr. John T. T. Hundley as Chairman, was appointed to work with the State Board of Medical Examiners in connection with medical licensure, and particularly the problem of providing standards under which the most competent of the large group of foreign graduates now in this State can be admitted to the examinations given by the Board and to licensed practice upon passing the examinations.

The solution of this problem was made difficult because the schools of graduation of these applicants have not been and cannot be inspected and evaluated by the grading agencies of the professional associations, and by reason of the obviously inadequate medical training given by schools in continental Europe during the past ten years.

Dr. Hundley's committee, working in co-operation with a like committee from the Board of Medical Examiners, gave the problem a great deal of study, and finally, with the assistance of the attorney for the Society, prepared tentative drafts of proposed bills to make a new plan of licensure effective as to these foreign graduates. Copies of these drafts were sent to all members of Dr. Hundley's committee, and the Legislative Committee and to the members of State Board of Medical Examiners, for study and comment. Thereafter a joint meeting of Dr. Hundley's committee and the Examining Board was held in Richmond, at which meeting a number of changes were made in the drafts which had been prepared. At a still later time Dr. Hundley met with the Legislative Committee, at which time some further changes were made, and four separate bills approved for introduction in the General Assembly.

Senator Button, of Culpeper, a very able member of the Senate of Virginia, united with your Chairman and Dr. Caudill as patrons of these bills. They were referred to the Senate Committee on General Laws, were promptly reported by the Committee, and later passed the Senate without opposition. In the House of Delegates they were referred to the House Committee on General Laws, but no action was taken by this committee for several weeks. At Dr. Elliott's suggestion they were referred to a sub-committee for study, and after this sub-committee had brought in a favorable report, all four bills were reported unanimously by the full committee and went to the House for final consideration. No real opposition developed, and during the last week of the session they were passed without a dissenting vote. This was particularly pleasing to the Board of Medical Examiners as the main bill, which deals with licensure of foreign graduates, required a four-fifths vote to make it effective in time for the June examinations. The bills have been signed by Governor Stanley and are now a part of the medical statutes.

The amendments to the licensing statutes authorize the Board of Medical Examiners to admit to the examinations any graduate of a foreign school which has not been inspected and approved if the Board is satisfied that the candidate's postgraduate training and experience or his professional education, supplemented by two years of internship training in the United States or Canada, is of such duration and character that his qualifications are equal to those required of other candidates. The two years of internship training in this country or Canada must be in a hospital approved for such purpose by the Council on Medical Education and Hospitals of the American Medical Association. One year of postgraduate study in an approved American school may be substituted for one year of internship training. The purpose here is not to open the examinations to incompetent and untrained men, but to those whose competency has been demonstrated by other evidence than inclusion of their professional schools on the A. M. A. approved list. No graduate of any school in this country which does not meet A. M. A. standards can qualify under the amended statutes.

THE RESPONSIBILITY IMPOSED ON THE BOARD OF EXAMINERS.

While the Legislative Committee believes that the licensing statutes as now amended are satisfactory, and are in line with those of other States, it realizes that a lax administration of the new licensing requirements by the Board of Examiners can result in the admission of a large number of foreign graduates to Virginia practice whose basic medical education is not of that high quality obtained in Grade A schools in this country. It is well known that for the decade following 1940 the medical schools in continental Europe have been inadequately staffed and poorly equipped, and it is this situation that makes the two-year internship or postgraduate work in this country essential. Unfortunately, the teaching load in our own schools is too heavy to permit postgraduate work for many of these candidates, and this leaves hospital training as the basic supplemental requirement for preparing them for practice. If this training is perfunctory, or is not properly supervised by an interested hospital staff, then the candidates will not be prepared for the examinations or for the practice of medicine.

At one of the preliminary committee meetings Dr. John B. Truslow, Dean of the Medical College of Vir-

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ginia, suggested that the Board require from the approved hospital a fully detailed report on and an evaluation of the candidate's work therein during his residency or internship. If the Board requires and can obtain reports of this nature on these men, and will refuse to open the examinations to candidates whose work does not measure up to high standards, it should be able to confine licensure to applicants whose competency is fully established. The discretion given the Board under the amended statute constitutes a challenge and imposes a responsibility which this Committee is confident it can and will meet wisely. If, however, experience demonstrates that incompetent and untrained men are taking the examination under the statute, it should be repealed in 1956 and a new solution found for the problem.

OTHER CHANGES IN THE LICENSING STATUTES.

Other changes in the statutes may be of interest. Under the law prior to these amendments professional schools were required to register with the State Board of Education as having met prescribed standards, which Board then certified the schools to the Examining Board. Hereafter the Examining Board will get its gradings directly from the agencies which inspect and grade the professional schools. This change has long been favored by the State Board of Education. Applicants for the examinations must now be citizens of this country, or have filed a declaration of intention to become citizens. Before taking the examinations evidence of indentity of each applicant is required. Supporting documents in foreign languages must be translated. Reciprocal licensing with foreign countries is no longer permitted. Some increases in the fees for examinations and licensing are authorized, the present fees, which have not been changed for many years, not producing sufficient revenue to meet the expenses of the Board.

Under the provisions of Section 54-276.7 the Board was authorized to grant certificates to certain Federal medical officers permitting them to engage in private practice. As certain of these officers cannot meet the licensing requirements of this State, the Board preferred to give up the authority (which it had never used) rather than to be importuned to grant certificates to men it considered not fully qualified. This change in the law was made at the request of the Examining Board.

The sections defining unprofessional conduct and prescribing the grounds for refusing admission to the examinations and for the suspension and revocation of licenses were rewritten to include in such grounds insanity, engaging in practice under a false name, and impersonating another physician, and to clarify the provisions and place them in more logical order.

APPEALS IN SUITS AGAINST THE BOARD.

During the past seven or eight years a number of candidates who had been refused admission to the examinations by reason of their inability to meet statutory requirements have petitioned the courts to require the Board to open the examinations to them and to award them licenses upon their making passing grades. Unfortunately several of these suits were decided against the Board, and under the law the decision of the circuit or corporation court hearing the matter was not subject to appeal. Although the Board has been successful in defending the last three suits of this nature instituted against it, its members are still of the opinion that the right of appeal should be available to the losing party in suits of this character. The amendment permits appeals in such cases, and thereby conforms the practice to that applicable to similar suits brought against other examining boards. The amendment should eventually change the present confusing situation in which different trial courts reach diametrically opposed but nevertheless final decisions on the same state of facts.

BOARD OF EXAMINERS FOR OPTICIANS.

While the foregoing changes in what is usually called the Medical Practice Act came about through bills sponsored by this Committee and introduced by its Chairman and Dr. Caudill, the General Assembly enacted other legislation which directly touches our profession and which should be noted here. At the annual meeting of the Society last October a resolution was adopted approving the creation of a Board of Examiners for Opticians, and your Committee felt obligated to work for the passage of a bill which was introduced for that purpose. The bill became law, and under its provisions the Virginia State Board of Opticians was created to license and regulate this practice. The Board will consist of two physicians who practice Ophthalmology, two Opticians, and one Optometrist. Each physician member will be appointed by the Governor from a list of three names submitted by The Medical Society of Virginia.

SPECIAL LICENSING BILL DEFEATED.

About a year ago the Circuit Court of Fairfax County sustained the Board of Examiners in refusing to admit Dr. Leslie Upton, a graduate of Middlesex University School of Medicine, of Waltham, Massachusetts, to the examinations. At the recent session of the General Assembly a bill was introduced which if passed would have required the Board to give the examinations to Dr. Upton and to award him a license upon making passing grades. This was the same kind of special bill which Mr. Stuart Carter got through the Assembly in 1952 for the benefit of Dr. Smith Taylor, another graduate of a sub-standard school. Dr. Taylor has never taken the Virginia examination, and the Upton bill was killed in the General Laws Committee of the Senate.

There were many other matters which demanded the time and thought of the legislative members of the committee during a busy session, but most of these were well covered by the press and will not be discussed here.

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We again express our appreciation of the splendid support given us by loyal members of the Society all over our State.

Respectfully submitted,

JAMES D. HAGOOD, M.D., *Chairman*
W. C. CAUDILL, M.D.
W. C. ELLIOTT, M.D.
DEAN B. COLE, M.D.
CARRINGTON WILLIAMS, M.D.
J. EDWIN WOOD, JR., M.D.
FRANK A. FARMER, M.D.
M. S. FITCHETT, M.D.
WALTER P. ADAMS, M.D.

AMERICAN MEDICAL EDUCATION FUND

The Time is Now

A sense of security is a dangerous thing—especially when that security is of the false variety. One of the truly great blessings of living in this nation is the blessing of freedom. And because we have always known freedom, we quite naturally have succumbed to the belief that security would always be ours.

Only now are we awakening to the fact, distasteful though it may be, that there are those who would use our feeling of security as a weapon to destroy completely the very freedom upon which this great nation has been built. They know that to feel secure is a sure way to lose the zest of combat, to refuse the challenge, to dull initiative. They know that it is one sure way to socialism, a way of life from which few return.

The medical profession is today spearheading the fight to keep this country free and to arouse a long sleeping public to the understanding that it must shake off the lethargy of two decades and gird itself for battle. The lay public may not understand technical jargon, but it does recognize leadership.

Leadership of the profession is manifested in the AMERICAN MEDICAL EDUCATION FOUNDATION. The Foundation, realizing that medical education is clinging desperately to its last thin thread of independence, is proving that the "best defense is a good offense." The Foundation, refusing merely to fight a delaying action, is moving forward—unawed by the dark threat of government subsidy.

How well the Foundation is moving forward can be seen in that nearly $2½ million were raised during 1953 for the nation's 79 medical schools. This sum was raised through contributions by practicing physicians or business firms which contributed through the National Fund for Medical Education. Despite the fact that this sum represents but a fifth of the total actually needed, it has kept at least one school from closing its doors, and has enabled others to establish new departments, obtain new faculty members, etc.

As of July, 1953, the Foundation, in cooperation with the National Fund, has distributed nearly $5,000,000 to the 79 schools. And that means that the first round has been won. But—battles such as this are not won in the first round, and frankly, this one has all the earmarks of a long term struggle. However, there is every reason to believe that *if every* physician does his part, the medical schools of America will still be standing free and resolute at the end.

Contribute now to the A.M.E.F.

ROBERT I. HOWARD, *Exec. Secretary*
The Medical Society of Virginia

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Medical Society of Virginia Cancer Committee

Chairman, George Cooper, Jr., M. D.

Medical School Building, University, Va.

Reprints of this and preceding Bulletins may be obtained from this office

May 1, 1954

Summary of
Present Knowledge on Smoking in Relation to Lung Cancer

(Excerpts from a Recent Statement by the American Cancer Society)

The present evidence concerning a possible relationship of smoking to lung cancer comes mainly from three sources:

1. Mortality Reports and Studies of Time Trends

The age standardized death rates for most sites of cancer have not changed greatly during the last several decades. Male death rates have declined somewhat for cancer of the stomach, skin and liver but have increased somewhat for cancer of the prostate, intestine and pancreas. Female death rates have declined considerably for cancer of the stomach and liver and have increased somewhat for cancer of several sites including ovary and pancreas. However, the single dramatic change is in the standardized death rate of lung cancer among males which rose from 3.6 per 100,000 population in 1930 to 19.5 in 1950.

During the period of time in which lung cancer death rates have been increasing, a great many changes have taken place in our environment and habits of life. Among other changes, there has been a tremendous increase in the use of motor fuel and fuel oil and the per capita consumption of cigarettes. Cigarette smoking as an almost universal habit began some years earlier among men than among women. There has been little change in the consumption of coal in the United States.

2. Laboratory Evidence.

Recently, laboratory workers have succeeded in producing cancer by applying material condensed from cigarette smoke to the skin of mice. Experiments are now under way to determine what ingredient of cigarette smoke is responsible for this effect. Although these experiments show the presence of a carcinogen, or cancer producing agent, in material condensed from cigarette smoke, no one has yet succeeded in producing epidermoid carcinoma of the lung in mice by exposing them to cigarette smoke. (Most lung cancer in man is of the epidermoid type.) The occurrence of adenomas of the lung has been increased by exposing genetically susceptible rabbits to cigarette smoke.

It is known that soot from coal can produce skin cancer both in man and in experimental animals. It has also been shown that fumes from fuel oil and exhaust fumes from automobiles contain considerable quantities of chemical substances which will produce cancer when smeared on the skin of a mouse.

The laboratory experiments seem to indicate that some chemicals capable of producing cancer must be applied for a considerable proportion of the normal life span of an animal in order to have this effect. Therefore, there is some reason to suppose that a period of many years (perhaps 15 to 40 years) of exposure might be required for a chemical substance to produce lung cancer in man.

3. Smoking Habits in Relation to Lung Cancer.

A number of investigators have compared the histories of smoking as told by lung cancer patients with the histories of smoking as told by samples of people who do not have lung cancer. In all such studies, a larger percentage of people with a history of heavy cigarette smoking has been found among the lung cancer patients than among the controls. However, the results of these studies differ widely in the degree of association found between lung cancer and history of cigarette smoking.

Although the details varied, essentially the same design was used in all of these studies and many statisticians have doubts as to the validity of this method of obtaining information. Whether or not the general finding of an association between smoking and lung cancer is valid, the degree of association is in doubt because of variations in the findings of different investigators.

Discussion

Although no one of the three types of evidence just described would be sufficient in itself as a basis for positive conclusions, all three taken together create suspicion that cigarette smoking does increase to some degree the probability that an individual

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will develop lung cancer. More evidence is needed to establish the degree of the danger, if it is proved to exist.

At the present time, the possibility cannot be ruled out that air pollution from coal and oil fumes is also a factor in the increase in death rates from lung cancer. It is also possible that human lung cancer results from a combination of factors, including smoking and exposure to soot and fumes from coal, oil, and motor fuels.

Constitutional susceptibility — as yet only theoretical — may also favor the occurrence of lung cancer, as it appears to do for some cancers of other sites.

Conclusions:

1) Evidence to date justifies suspicion that cigarette smoking does, to a degree as yet undetermined, increase the likelihood of developing cancer of the lung.
2) Available evidence does not constitute irrefutable proof that cigarette smoking is wholly or chiefly or partly responsible for lung cancer.
3) The evidence at hand calls for extension of statistical and laboratory studies designed to confirm or deny a causal relationship between cigarette smoking and lung cancer.

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WOMAN'S AUXILIARY TO THE MEDICAL SOCIETY OF VIRGINIA

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President-Elect ..... MRS. MAYNARD EMLAW, Richmond
Recording Secretary ..... MRS. LEE S. LIGGAN, Irvington
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MRS. LEMUEL E. MAYO, Portsmouth
Treasurer ..... MRS. WILLIAM C. BARR, Richmond
Publication Chairman MRS. WM. S. GRIZZARD, Petersburg

Auxiliary News.

DANVILLE-PITTSYLVANIA ACADEMY

The Food Sale held by the Auxiliary on March 5 at the Parish House in Mt. Vernon Methodist church in Danville was quite successful. The sale was held by members of the Auxiliary to raise funds for their Nurses Scholarship Fund (two scholarships are being offered to nurses who wish to further their training in graduate study for six months). The nurses then come back to Memorial Hospital for duty in Danville for a year. The committee working with the Auxiliary in this project was headed by the Ways and Means Chairman, Mrs. M. H. McClintic.

Each member of the Auxiliary supplied food for the sale. It was so successful that we expect to have another sale this Spring.

On Monday, March 22, the Auxiliary had a White Elephant sale for members. Each one furnished two gifts to be auctioned off by Mrs. John L. Clare and Mrs. John W. Hooker. The sale was held in the home of the Auxiliary President, Mrs. Charles A. Easley, Jr. Tea was served. Proceeds from the sale were added to funds for the Nurses Scholarship Fund.

(MRS. DRAKE) SALLY W. PRITCHETT,
Publicity Chairman.

ARLINGTON

The Woman's Auxiliary of Arlington held their annual Fashion-show and luncheon, Tuesday, March 9th, at the Washington Golf and County Club, for the benefit of Arlington hospital. Proceeds realized will be applied to the balance due on the Doctors' "Call Board", recently installed by the Auxiliary in the hospital. Fashions were by Lady Hamilton, Inc. of Arlington. There was a capacity crowd and all in gay attire. Numerous merchants in Arlington donated door prizes and flowers for this festive occasion. Mrs. M. W. Glover, Chairman, and her committee are to be congratulated for the success of this affair. Music was furnished by the Ashton Heights Womans Club (Arlington, Va.), who turned the check over to the hospital also.

The Woman's Auxiliary has been busy with details in connection with helping the Doctors publicize the AAPS Essay Contest. The response has been very satisfying and the prizes offered by the County Medical Society are an inducement to have the high school student participate. Together with the national and county prizes, the students are eligible for a total of $1,675.00.

Our congratulations to Dr. and Mrs. A. R. MacPherson on the birth of a bouncing boy, "Archibald", recently.

Dr. W. C. Welburn reports that Mrs. Welburn is recuperating nicely from injuries received in an automobile accident while vacationing in St. Petersburg, Fla. recently. Mrs. Welburn is one of the oldest members to the Arlington Auxiliary and all of her friends will be happy to hear that she is expected home in May.

The Woman's Auxiliary of Arlington celebrated Doctors Day on March 30th with radio and television programs on the newscasts throughout the day. March 29th, the Arlington Chamber of Commerce very graciously gave their entire half-hour program to the Auxiliary for the purpose of celebrating Doctors Day. Mrs. Robert H. Mitchell, Publicity Chairman, acted as moderator and also gave the history of this day. Mrs. Lee B. Martin, President, presented the purpose and the beginning of the Arlington Chapter of the Auxiliary. Mrs. Sigmund Newman presented some of the good deeds by the Auxiliary for the benefit of the Arlington Hospital. Mrs. Robert

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H. Detwiler acquainted the radio audience with the philanthropic activities of the Auxiliary. We were very happy to have as our guest on this program, Dr. J. Raymond B. Hutchinson, President of the Arlington County Medical Society, who gave a very informative talk and then answered questions asked by Mrs. Mitchell, to better inform the listening audience regarding the Medical Society's purpose in the County. Also for Doctors Day, the members of the Auxiliary collected drug samples from the Doctors offices and gave them to the Arlington Hospital. There was newspaper coverage for this day also, and a picture of Mrs. Martin and Dr. Hutchinson in the paper.

E. MITCHELL
(MRS. ROBT. H. MITCHELL)
Publicity Chairman

NORFOLK

The March meeting of the Norfolk Auxiliary was a luncheon at the Norfolk Yacht and Country Club honoring the State President, Mrs. K. W. Howard. Mrs. Maynard Emlaw of Richmond, president-elect was guest speaker, speaking on "Why an Auxiliary?"

Mrs. Emlaw stated as a most important purpose of the auxiliary "to extend the aims of the medical profession to all organizations which work toward the advancement of health and health education."

The auxiliary works also "to cultivate friendly relations and promote mutual understanding among physicians' families; to contribute to any endeavor on the request of our medical society, and to assist in the entertainment at all conventions participated in by our medical society.

"For certainly," Mrs. Emlaw continued, "no other group of women share more mutual problems than doctors' wives and they should serve to knit us more closely together.

"The auxiliary is the one organization where we have a complete understanding of each other's difficulties—the late meals, the broken social engagements, and the like."

Mrs. J. R. St. George, president of the Woman's Auxiliary to the Norfolk County Medical Society, announced as its Spring project a luncheon April 21, when Dr. Walter B. Martin, president-elect of the American Medical Association, as speaker, will explain the president's proposed program.

A meeting for the election of officers will precede the luncheon.

Mrs. St. George called upon members to work with the doctors on the public medical forum, which they are planning.

PETERSBURG

The Petersburg Medical Auxiliary honored the doctors by sending to each one a red carnation to be worn on Doctor's Day. Mrs. Garnett Link and Mrs. Francis Payne, Jr., were in charge.

The March meeting of the Auxiliary was held at the greenhouse of a local florist. Each year the members are asked to bring a container and cut flowers are invited to see the Spring flowers. They provided to make arrangements. Cokes and cookies are served. This meeting is looked forward to each year with much pleasure.

MARGARET S. WHITTLE
(MRS. JOSEPH)

NEWPORT NEWS-WARWICK

The Woman's Auxiliary to the Newport News-Warwick County Medical Society held its regular luncheon meeting Wednesday, March 24, at 12:30 at the home of Mrs. Russell Buxton on Shore Drive, Christopher Shores. There were thirty-five members present.

Mrs. K. W. Howard of Portsmouth, president of the Woman's Auxiliary to The Medical Society of Virginia, was the guest speaker. Mrs. Howard outlined the work being done by the various auxiliaries in the state.

Mrs. M. R. Emlaw of Richmond, president-elect of the Woman's Auxiliary to The Medical Society of Virginia, was also guest at the luncheon.

Mrs. T. W. Caldroney reported the progress being made in the future Nurses Club.

A contribution of $75.00 was made to Patrick Henry Hospital for the chronically ill in commemoration of Doctor's Day, March 30. The money will be used to purchase oxygen, intravenous and transfusion equipment at the hospital.

BESSIE G. AMORY
(MRS. GUY C.)

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EDITORIAL

Blue Cross-Blue Shield Coverage of Mental Illness

DOCTOR SPESSARD'S article in the April issue of the "Monthly" is thought provoking. It should stimulate Blue Cross-Blue Shield people and others who are interested in the adequacy and soundness of non-profit Plans to review the programs with which they are connected and determine whether or not their Plans are keeping up with the changes and accomplishments of psychiatric medical practice. Because recent successes in the field of mental health are so widely recognized, it is all the more important that the still existing limitations in Blue Cross-Blue Shield services available to the mentally ill be correctly interpreted and fully understood by the medical profession as a whole. The progress made by Blue Cross-Blue Shield has been no less phenomenal than the progress in psychiatry, but, as does psychiatry, Blue Cross-Blue Shield has problems that must be overcome before its development along certain lines can be continued.

Psychiatry and Blue Cross-Blue Shield are working toward the same end, the complete well-being of the American public. They both are "modalities", to borrow a term from physical medicine, for the promotion of individual health and of public health. The physicians, the hospital people, and also the public representatives who serve on the Boards of Blue Cross and Blue Shield Plans are quite aware of the unmet needs of those who are mentally ill. As do psychiatrists, Blue Cross-Blue Shield Board members deprecate the continuance of the general public's attitude and dark-age concept that mental illness is related to demoniacal possession. As will be discussed, this unfortunate attitude is impeding their work toward promoting individual and public health in the same way that it makes things more difficult for psychiatrists. It is not misconception concerning mental illness that deters Blue Cross-Blue Shield from increasing further its coverage of services to the mentally ill. There are, however, problems of more specific and practical nature that are deterrents. It is fundamental that Blue Cross-Blue Shield must "sell" and must be able to deliver what it sells. As concerns care of mental illness, Blue Cross-Blue Shield, in many areas of the country, still encounters difficulties in meeting these basic requirements of Plan development.

Blue Cross-Blue Shield, guided by the physicians who serve as its Directors, could devise a health service program which would include only those services generally deemed advisable by the profession but which, nonetheless, would cost a family as much as $20.00 a month. A rate of that magnitude, of course, would preclude success in enrolling those people of borderline economic status who actually need such a program the most. In the case of services, just as in the case of commodities, successful selling is dependent upon the ability to pay of the potential customer. Accordingly, before Blue Cross-Blue Shield will be able to include more psychiatric services, psychiatry must have further success in devising more rapid, less expensive methods of effective care and must bring the total cost of such care within the reach of the entire Blue Cross-Blue Shield population. Because prepayment, to be truly effective, cannot fall far short of being payment in full and because prepayment cannot reduce the total of medical care expense, Blue Cross-Blue Shield is sincerely hopeful that the average expense of adequate psychiatric services will soon more closely approximate the average expense of other services it provides. At that time it will have greater chance of success in its attempt to sell psychiatric services.

Part of Blue Cross-Blue Shield's problem of selling is the problem of public attitude. Even if someone could afford a $20.00 a month comprehensive program, he

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would have to be educated to *want* to pay that much for it. Blue Cross-Blue Shield can do no more than provide the protection the public wants and is willing, as well as able, to pay for in full. Blue Cross-Blue Shield can help, and does help, practicing physicians in their endeavor to educate the public concerning medical care needs. But Blue Cross-Blue Shield would jeopardize its very existence were it to include services which, even though needed, were not requested by the public and for which the public would be unwilling to pay. And here is where the problem of attitude toward mental illness comes in. Because too many people still consider mental illness somewhat degrading and disgraceful they do not properly evaluate their chance of developing such illness. They accept the possibility of accidental injury and they accept the possibility of socially acceptable diseases and physiological dysfunctions. But they react ostrich-like to the possibility that emotional stresses and strains might become too much for them. They refuse to see themselves in need of psychiatric services; "Maybe some other person, but not me, and I don't want to pay for something I'm not going to use". The individual saying this, of course, is not a patient who has had occasion to receive psychiatric services; he is the so-called "average" individual to whom Blue Cross-Blue Shield attempts to sell its Contract.

There is ancther reason Blue Cross-Blue Shield has been developing slowly as concerns care of the mentally ill. Not only must it sell its "product", medical care services, but it must also be able to deliver. Quite understandably, a Blue Cross-Blue Shield subscriber expects, when occasion arises, to receive all of the services he needs that are listed in his Contract. Today, however, Blue Cross-Blue Shield could not possibly deliver adequate psychiatric services to every subscriber who might be in need of them. Considering only the relatively short-term psychiatric patients who can be materially aided, there are not enough psychiatrists or sufficient hospital facilities to make it possible for Blue Cross-Blue Shield to live up to a more inclusive Contract. Blue Cross-Blue Shield necessarily must synchronize its Contract not only with the services available in metropolitan medical centers but also with the services available in the average community. This average community supports a hospital of about sixty-five beds and is served by physicians in general practice, few of whom have been able to keep abreast with psychiatry's advances and developments.

As opportunity to sell and to deliver keeps increasing, Blue Cross-Blue Shield will continue to do as much as it can, as soon as it can, to assist in the care of mentally ill subscriber-patients. There has already been notable progress. Twenty years ago but very few of the community hospitals which developed and effected the unique Blue Cross idea were willing to admit psychiatric patients. At that time, care of the mentally ill was generally considered a responsibility of government. Accordingly, psychiatric services were almost completely left out of the first Blue Cross hospital programs. But psychiatry started doing things, and its accomplishments made hospital folks take notice. The idea developed that the State should continue to provide long-term custodial care and that, whenever possible, local communities should provide diagnostic and therapeutic facilities and definitive care of short-term cases. The American Hospital Association, as well as the National Association for Mental Health, for many years has encouraged general hospitals to provide care of the mentally ill, and as more general hospitals developed psychiatric service facilities, Blue Cross increased its provisions concerning these services.

Today, 22% of the Blue Cross Plans in the United States provide the same benefits to the patient for mental illnesses as for other illnesses, when he is admitted to a Blue Cross hospital. These Plans cover about 10 million persons. Also, 40% of the Plans,

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covering about 19 million people, provide a somewhat less complete coverage such as regular benefits for a limited number of days, the number of days within the limit, however, usually being 50% to 100% greater than the average number of days hospital stay required by subscriber-patients with organic disease. Accordingly, a total of 62% of the Blue Cross Plans, with a total membership of 29 million, provide fairly substantial benefits for patients with mental illness. Another 8% of Blue Cross Plans offer patients regular benefits for the length of time required to rule out possible existence of organic disease as the basis of the complaints presented.

The present-day status of Blue Cross-Blue Shield coverage of mental illness reflects noteworthy accomplishments during the past twenty years, which in turn have been based upon the accomplishments of psychiatry in reducing the expense of adequate care, in making such care more readily available, and in educating the public to want provisions for care of mental illness. As psychiatry enjoys further success in these areas of its endeavor, Blue Cross-Blue Shield will keep pace by further development of its coverage of mental illness. The future looks bright.

RICHARD J. ACKART, M.D.
Executive Director
Virginia Medical Service Association

SOCIETIES

The Williamsburg-James City County Medical Society

Had their April meeting as Ladies and Guest Night on the 7th. Cocktails were served at the home of Dr. and Mrs. J. E. Barrett, followed by dinner in the Eastern State Hospital Staff dining room. The speaker on this occasion was Mr. Norman Marshall whose subject was Apothecary of Colonial Williamsburg.

Danville-Pittsylvania Academy of Medicine.

At the regular monthly meeting of the Academy on February 10, Dr. Paul Bunce, of the Department of Urology at the University of North Carolina School of Medicine, spoke on the Treatment of Cancer of the Prostate.

At the meeting on March 12, Dr. Eric Schelin of the Medical College of Virginia, Richmond, spoke on Carcinoma of the Cervix.

Dr. J. W. Hooker and Dr. Jefferson D. Beale, Jr., both of Danville, are president and secretary, respectively of the Academy.

The Fourth District Medical Society

Met in Farmville, on the afternoon of April 13 in conjunction with the Staff of the Southside Community Hospital. The President, Dr. A. Tyree Finch of Farmville gave his address. Dr. John Guerrant of the University of Virginia Medical School spoke on Pulmonary Functions in Certain Lung Diseases and Dr. Thomas H. Hunter, also of the University, spoke on Some Factors Involved in the Choice of Anti-Biotic Therapy. Following a short business meeting by the Society, Cocktail Hour was held at Hotel Weyanoke, followed by dinner at the Hotel as guests of the Hospital Staff.

Roanoke Academy of Medicine.

The meeting of the Academy on April 5, was conducted by the Staff of Gill Memorial Hospital in the Ballroom of Hotel Roanoke at 8:00 p.m. Speakers on this occasion were Dr. Paul Hawley, Director, American College of Surgeons, on "Professional Freedom in Medical Practice", and Dr. Winchell McK. Craig, Professor of Neurosurgery of the Mayo Clinic. His address was on 'Intraspinal Tumore, and the Part They Play in General Diagnosis".

The Warwick-Newport News Medical Society

Had its regular meeting on April 13 in the Coca Cola Building in Newport News. The speaker was Dr. A. R. Coppola of Newport News, whose subject was "Neuro-Surgical Aids in Diagnosis". Refreshments followed.

Dr. William A. Read is president and Dr. T. Ashton Carmines secretary-treasurer.

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NEWS

Calendar of Coming Events

Virginia Academy of General Practice—Richmond, May 4-7

Virginia Society of Ophthalmology and Otolaryngology—Old Point Comfort, May 7-8

Virginia Academy of Science—Charlottesville, May 13-15

American Gynecological Society — Hot Springs—May 20-22

Virginia Heart Association—Hotel Jefferson, Richmond—June 13.

American Medical Association — Annual Meeting—San Francisco, June 21-25

American Congress of Physical Medicine and Rehabilitation—Washington, D. C., September 6-11

The Medical Society of Virginia (Annual Meeting)—First Interstate Scientific Assembly—Hotel Shoreham, Washington, D. C., October 31-November 3.

Session on Legal Medicine at San Francisco.

In recognition of the growing importance of the many situations in which medicine may contribute to a clarification of medicolegal issues and of the interest and concern of physicians in such situations, there will be presented at the San Francisco meeting of the American Medical Association in June a Session on Legal Medicine in the Section on Miscellaneous Topics of the Scientific. This Session will be held under the immediate sponsorship and direction of the Committee on Medicolegal Problems which has arranged an informative program to include discussions on topics of practical value to practitioners who, whether they like it or not, will some day become personally concerned in a medicolegal involvement. The following papers will be presented:

Advice to the Medical Witness—W. I. Gilbert, Esq., President, Los Angeles Bar Association

Malpractice, an Occupational Hazard—Louis J. Regan, M.D.

Medicolegal Problems Related to Sterilization, Artificial Insemination and Abortion—J. W. Holloway, Jr., Esq., and Edwin J. Holman, Esq.

Prevention of Transfusion Accidents—Alexander S. Wiener, M.D.

Legal Aspects of Medical Partnership—George E. Hall, Esq.

Trauma, Stress and Coronary Thrombosis—Alan R. Moritz, M.D.

This Session represents a practical and somewhat new approach to a solution of some of the situations in the medicolegal field that have caused, or that in the future may cause, uncertainty and possible embarrassment on the part of the physician. The program has been carefully arranged with that objective in mind and a physician will find much of value in the six papers. The meeting will be held on Thursday morning, June 24, in the White Room of the Masonic Temple located at 25 Van Ness Avenue and will begin at 9:00 a.m. and conclude at 12:00 noon.

The Virginia Society of Ophthalmology and Otolaryngology

Is holding its annual meeting at the Chamberlin Hotel, Old Point Comfort, Virginia, on May 7 and 8. There will be recognition of members associated with the Society for twenty-five or more years. Dr. Fletcher Woodward of Charlottesville will be the guest of honor and Dr. Banks Anderson of Duke University, Durham, N. C., will be the guest speaker. His subject will be "Ocular Manifestations of Systemic Disease".

Dr. Peter N. Pastore and Dr. L. Benjamin Sheppard, both of Richmond, are president and secretary of the Association, respectively.

The Virginia Pediatric Society

Held a most interesting meeting at the Homestead Hotel, Hot Springs, Virginia, on Febraury 26 and 27, under the presidency of Dr. T. Stanley Meade of Richmond. The guest speakers who were on both dates of the meeting were Dr. Joseph Stokes, Jr., of the Department of Pediatrics of the University of Pennsylvania, Philadelphia, Dr. Lawson Wilkins, Associate Professor of Pediatrics, The Johns Hopkins Hospital, Baltimore, and Dr. Waldo E. Nelson, Temple University and St. Christopher's Hospital for Children, Philadelphia. Members of the society who presented papers were Dr. Ralph Ownby of the Medical College of Virginia, and Dr. James B. Wood, Jr., of the University of Virginia.

Several exhibitors at the meeting arranged for social events for the doctors and their wives.

Eleven new members were elected and the follow-

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ing officers were named: President, Dr. T. J. Humphries of Roanoke; vice-president, Dr. F. Read Hopkins of Lynchburg; and secretary-treasurer, Dr. A. Page Booker, Charlottesville, succeeding Dr. William Grossmann of Petersburg.

A Conference on Therapy

Was held at the University of Virginia on April 23. At the morning session, Management of some Phases of Cardiovascular Disease was discussed, and after lunch, the subject was Management of Obstetric and Gynecological Problems. In addition to several speakers from the two departments at the University, the guests were Dr. Edward D. Fries, Adjunct Professor of Medicine at Georgetown University School of Medicine, Washington, and Dr. H. Hudnall Ware, Jr., Professor of Obstetrics and Gynecology at the Medical College of Virginia, Richmond.

To Be Honored by Medical College of Virginia.

Dr. James Loving Hamner, rural practitioner of Mannboro, former president of The Medical Society of Virginia and a member of the State Board of Health, is one of two alumni who will receive honorary degrees at the Medical College of Virginia Commencement on June 1.

The other doctor to be honored is Dr. Randolph Lee Clark, Jr., of the class of '32, prominent surgeon of Houston, Texas.

Inaugural Address to Be Broadcast.

Even if you cannot attend the American Medical Association's 103rd Annual Meeting, June 21-25, you will be able to hear the president's inaugural address broadcast directly frmo the ballroom of the Palace Hotel in San Francisco. The remarks of President-Elect Walter B. Martin will be broadcast on a nationwide radio network Tuesday evening, June 22. The program will be telecast locally. Immediately following the broadcast, a reception and ball honoring Dr. Martin will be held.

The Virginia Academy of General Practice

Is holding its fourth annual scientific assembly at the Jefferson Hotel, Richmond, May 5, 6 and 7. On the 5th, the Virginia Chapter, American College of Chest Physicians meets in the morning, and the Virginia Diabetes Association will have its session in the afternoon. The Virginia Academy of General Practice will have its meeting of the Board of Directors that evening and the scientific sessions on the following days. Attractive entertainments have been arranged for the members and for the ladies accompanying them. A number of technical and scientific exhibits will add to the interest of this meeting.

The American Congress of Physical Medicine and Rehabilitation

Will hold its annual scientific and clinical session September 6-11, 1954 inclusive, at the Hotel Statler, Washington, D. C. All sessions will be open to members of the medical profession in good standing with the American Medical Association.

In addition to the scientific sessions, annual instruction seminars will be held, open to physicians as well as to therapists who are registesed with the American Registry of Physical Therapists or the American Occuptional Therapy Association.

Full information may be obtained by writing to the executive offices, American Congress of Physical Medicine and Rehabilitation, 30 North Michigan Avenue, Chicago 2, Illinois.

The Annual Essay Award contest, sponsored by the Congress, will be open to graduate students in the pre-clinical sciences and graduate students in physical medicine and rehabilitation. Manuscripts must be in the office of the Congress not later than June 1, 1954. For details, write to the executive offices.

News from State Health Department.

Dr. T. H. Valentine, medical director of the Brunswick-Greensville-Mecklenburg Health District, died April 3. Dr. Valentine was the oldest public health official in point of service in Virginia. He will be greatly missed in the public health program of the State.

Dr. R. D. Wallace has been appointed Director of the Bureau of Dental Health of the State Health Department. He will begin his service on May 1. Dr. Wallace is replacing Dr. W. H. Rumbel who died last November.

The J. Shelton Horsley Memorial Lectureship,

Established by Dr. Guy W. Horsley in 1947 in honor of his father, each year presents an outstanding speaker in the field of Surgery at a stated meeting of the Richmond Academy of Medicine in April. The address this year was given on April 13 by Dr.

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George T. Pack, Clinical Professor of Surgery at New York Medical College and attending Surgeon-in-Chief of Gastric and Mixed Diseases at Memorial Hospital, New York. His subject was "Management of Pigmented Moles and Malignant Melanoma."

Two Recipients of the Markle Fund.

Among the twenty-five appointments of faculty members of medical schools in the United States and Canada to be granted recognition for their research work by the Markle Fund are two from Virginia Medical Schools—Dr. Robert Q. Marston at the Medical College of Virginia, Richmond, and Dr. Oscar A. Thorup, Jr., of the University of Virginia School of Medicine, Charlottesville. The grant is for $6,000 annually for five years to the schools named where the doctors will teach and carry on their research. Dr. Marston's interest is internal medicine, and Dr. Thorup's is hematology and cardiology.

The Virginia Heart Association

Will meet at the Jefferson Hotel, Richmond, on May 13, at 2:30 p.m. The speaker is to be Mr. Blake Cabot, author of "The Motion of the Heart—the Story of Cardiovascular Research", whose topic will be "Telling the Cardiovascular Research Story".

Under the auspices of American Heart Association, Mr. Cabot visited and talked with hundreds of research scientists in hospital and medical-school laboratories throughout the United States. He will relate some of the behind-the-scenes accounts of his observations, which afforded intimate close-up views of how experiments are conducted, how the scientists learn from them, and how this new knowledge applies to the treatment of cardiovascular diseases.

Postgraduate Credit for Academy Members.

The American Academy of General Practice has given official sanction by its Commission on Education for *Formal Postgraduate Credit* to Academy members who attend the first Interstate Scientific Assembly of The Medical Society of the District of Columbia and The Medical Society of Virginia. This is to be at the Shoreham Hotel in Washington October 31-November 3, 1954.

The National Tuberculosis Association

Will hold its 50th Anniversary Meeting on May 17-21 in Atlantic City. The Ambassador, Chelsea, and Ritz Carlton Hotels will be used.

Beginning Monday morning, May 17, there will be medical sessions on basic considerations in tuberculosis; nontuberculous pulmonary diseases; environmental considerations in tuberculosis; laboratory developments; surgery in the treatment of tuberculosis, and nonpulmonary tuberculosis, and panel discussions on the epidemiology of cancer of the lung, the treatment of tuberculosis in infants and children, and changing concepts and modern treatment of tuberculosis. There will also be a number of small seminars on various aspects of tuberculosis treatment and research.

The general theme of the anniversary meeting will be "The Challenge of the Future." There will be a number of general sessions, the first of which will be Monday afternoon when the program will be built around "The People Against Tuberculosis—in Medicine, in Government, in Voluntary Agencies."

Martha Jefferson Hospital Enlarged.

The Martha Jefferson Hospital, Charlottesville, has been enlarged by the addition of a new $1,105,000 wing which was dedicated on March 20. The new wing has 28 double rooms for patients, a kitchen, laundry, four operating rooms, new maternity department and nursery, and new facilities for the radiology department. Dr. T. H. Daniel, treasurer of the hospital's board of trustees, was the main speaker at the dedication.

The old wing which was completed in 1929 is to be completely remodeled.

Dr. D. Lane Elder,

Hopewell, mayor of that city for nearly thirty-four years, has announced that he will quit City Council when his present term expires September 1.

Addition to List of Licensees.

Dr. Jacob S. Rand, Far Rockaway, New York, was also licensed by the Virginia State Board of Medical Examiners at its examinations of December 28, and should be added to the list published in the February MONTHLY.

Graduate Lecturers at University of Virginia School of Medicine.

In addition to lectures listed in the February *Monthly,* an addition and a change are reported for May:

Thursday, May 6 at 5:00 p.m.—Dr. Stewart Wolf, Professor of Medicine at University of Oklahoma School of Medicine, on "Stress and Human Gastric Function", and

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Wednesday, May 12 at 8 p.m.—Dr. Paul Aebersold, Director of Isotopes Division, Oak Ridge (Tenn.) Institute of Nuclear Studies, on "Radioactive Isotopes: Cancer Research, Diagnosis and Therapy".

All Virginia physicians are welcome and are invited without a fee.

Dr. R. S. Griffith,

Ninety-three year old physician of Waynesboro, has been honored by the local firemen of the Waynesboro Fire Company by being given a plaque as a tribute to his long service. He is a veteran of the bucket brigade days, having first joined the fire fighting unit in the former Basic City in 1892.

For Sale:

40 bed, well equipped, small town hospital. Address Physician's Hospital, Incorporated, Warrenton, Virginia. (Adv.)

Doctor's Office:

Ideal location for physician, outside of Washington, in expanding community of Alexandria, Virginia. Complete 5-room house available for use as office in area needing services of a physician. Owners willing to assist doctor to get started. Telephone Mr. Charles Gerstein for appointment, TE-6-9378. (Adv.)

For Sale—

One Coreco Color Camera with x-rays, gynecology, body surface and specimen attachments. Same as new. Original cost $790. Will sell for $490. W. L. Davis, M.D., 87 Twenty-ninth Street, Newport News, Va. Telephone 2-4532. (*Adv.*)

OBITUARIES

Dr. H. Stuart MacLean,

Richmond, died April 2 in a local hospital, after having been in bad health for a couple of years. He was eighty years of age and came to this country from Inverness, Scotland, about the turn of the century. He graduated in medicine from Long Island College of Medicine in Brooklyn in 1895 and, shortly after completing his internship in Brooklyn, located in Richmond. With Dr. Robert C. Bryan, he founded Grace Hospital in Richmond in 1912, and was on its surgical staff until 1934. He was also chief medical officer of the Virginia Electric and Power Company, which position he held to time of his retirement. He served for a time on the faculty of the Medical College of Virginia and was a member of a number of medical organizations and was an elder in the Presbyterian church. His wife, a daughter and son and two grandchildren survive him.

Dr. Lomax Gwathmey,

For many years prominent surgeon of Norfolk and president of The Medical Society of Virginia in 1906, died March 22, at his home at Back Bay, near Virginia Beach, to which place he moved upon his retirement from active practice several years ago. Dr. Gwathmey was born November 5, 1869 and studied medicine at the University of Virginia from which he graduated in 1889. He also studied at Columbia University in New York and in Vienna and Heidelberg and on his return in 1895, he located in Norfolk where he became one of its prominent surgeons. In World War I he served in France and was invalided home in December 1918 and discharged early in 1919 with the rank of major. He was a past resident of the Norfolk County Medical Society and a member of various other medical organizations. His wife whom he married in August 1892 survives him.

Dr. Thomas Henry Valentine,

Lawrenceville, died April 3, shortly after he suffered a heart attack. He was seventy-five years of

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age and studied at the former University College of Medicine, Richmond, from which he graduated in 1901. He was perhaps the oldest public health official in point of service in Virginia, having served continuously in health work since 1908. Under his leadership, the health service had grown from a one room office in his section to a point where three counties in that section have eight nurses, four sanitation officers and five clerks. He was a Mason and Life Member of The Medical Society of Virginia. A daughter and son survive him, his wife having died in April 1953.

Dr. Charles R. Woolwine,

Health officer at Virginia Polytechnic Institute in Blacksburg from 1928 to 1951, died at a Richmond hospital March 19. Upon retirement, he had lived on a farm near Sealston in King George County. Dr. Woolwine was seventy-three years of age and a graduate of the Medical College of Virginia in 1908. He was engaged in practice with coal mine companies before locating in Blacksburg. He had for many years been a member of The Medical Society of Virginia. His wife and a sister survive him.

Dr. Louis S. Leo,

Norfolk, a specialist in eye, ear, nose and throat work and in plastic surgery, died March 17 in a Norfolk hospital, following a heart attack. He was fifty years of age and a graduate in medicine from the Medical College of Virginia in 1927. He later took postgraduate work at the University of Berlin and at the University of Vienna. He was a fellow of the American College of Surgeons as also of The Medical Society of Virginia and several other organizations. During World War II, he was a flight surgeon. He is survived by his wife and two daughters.

Resolution on the Death of Dr. Julius Dreher Willis

WHEREAS, it is with profound sorrow that the Roanoke Academy of Medicine records the death of its member and Past President, Dr. Julius Dreher Willis, which occurred on January 29, 1954, at the age of 68. We wish to extend to his family our deepest sympathy. His passing will not only be a great loss to his family, but also to the medical profession of this and surrounding communities, and to the numerous and genuine friends he has made through an active, devoted and fruitful career as physician, counselor, friend and civic spirited citizen.

BE IT RESOLVED, that a copy of this resolution be spread upon the minutes of this meeting of the Roanoke Academy of Medicine, and that copies be sent to the *Virginia Medical Monthly*, and to the American College of Physicians, and to the family.

IRA H. HURT, M.D.
S. BEVERLY CARY, M.D.
FRANK A. FARMER, M.D.
Committee.

Dr. Nathaniel F. Rodman.

The following resolutions were presented to the regular meeting of the Norfolk County Medical Society on March 1, 1954:

The death of Dr. Nathaniel Fulford Rodman, on January 14, 1954, deprived the Norfolk County Medical Society of a greatly beloved member.

Dr. Rodman, the son of the late Colonel and Mrs. William Blount Rodman, was born in Washington, North Carolina in 1891. His early education was received at Oak Ridge Institute and later at the University of North Carolina. Graduating from Jefferson Medical College in 1914, he took postgraduate training in the Presbyterian and Children's Hospital in Philadelphia and, soon after completing these internships, he accepted a commission in the United States Army Medical Corps where he served for the duration of World War I.

In 1918 Dr. Rodman returned to Norfolk and resumed the practice of medicine and surgery. This he continued until ill health caused him to retire, three years ago.

He was an active member of the staff of Leigh Memorial Hospital, De Paul Hospital and the Norfolk General Hospital. For years he was a member of the Executive Committee of the Norfolk General Hospital, serving diligently on many important committees. For a term of one year he was president of this hospital.

Dr. Rodman's greatest interest was in the Norfolk County Medical Society, serving as president in 1943-1944. He was also a member of The Medical Society of Virginia, the American Medical Association, and a fellow of the Southeastern Surgical Congress.

Dr. Rodman had a most attractive personality, being cheerful, honest, and conscientious. Modesty, sympathy and kindliness were other characteristics which endeared him to his many friends and patients. He had, at times, a decided opinion of his own which never interfered with his popularity, as he was tolerant of the ideas of others. He made many friends and most of them were permanent.

Flowers were his hobby. Many pleasant hours were spent in his garden.

WHEREAS the loss of Nat Rodman will bring great sorrow to his family, his friends and his patients,

BE IT RESOLVED, that this resolution be accepted and placed in the minutes of this Society, and,

BE IT FURTHER RESOLVED, that a copy of this resolution be sent to Mrs. Rodman.

(Signed) B. L. PARRISH
P. E. THORNHILL
A. B. HODGES, *Chairman*

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